This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

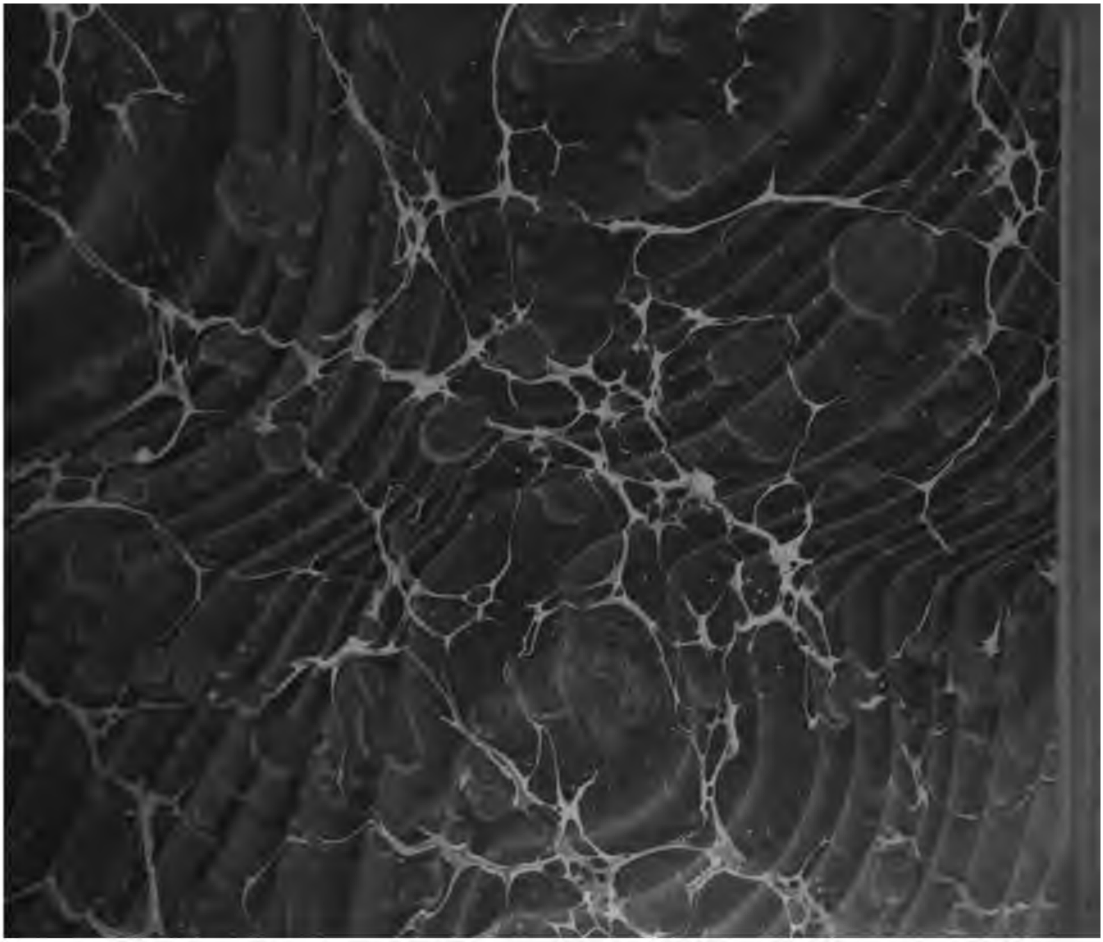
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
1885
LEVI COOPER LANE FUND



DISEASES

of the

INTESTINES.

IN TWO VOLUMES.

HEMMETER.

Vol. I.

By the Same Author

DISEASES OF THE STOMACH

Their Special Pathology, Diagnosis, and Treatment. Including Sections on Anatomy, Analysis of Stomach Contents, Dietetics, Surgery of the Stomach, etc.

Second Edition, Revised and Enlarged.
Colored and other Illustrations.

8vo.; 890 pages; Cloth, $6.00; Leather, $7.00; Half Russia, $8.00, net.

"We wish to express unqualified approval of the tendency which is shown to emphasize the simple and more practical methods of diagnosis."
—*New York Medical Journal.*

"The best contemporary treatise on diseases of the stomach which we possess, not only in America, but in the whole world."—*Prof. I. Boas, of Berlin.*

DISEASES
OF THE INTESTINES

THEIR SPECIAL PATHOLOGY, DIAGNOSIS,
AND TREATMENT

WITH

SECTIONS ON ANATOMY AND PHYSIOLOGY, MICROSCOPIC AND CHEMIC
EXAMINATION OF THE INTESTINAL CONTENTS, SECRETIONS, FECES,
AND URINE. INTESTINAL BACTERIA AND PARASITES; SURGERY OF THE INTESTINES; DIETETICS,
DISEASES OF THE RECTUM, ETC.

BY

JOHN C. HEMMETER, M.D., PHILOS.D.

PROFESSOR IN THE MEDICAL DEPARTMENT OF THE UNIVERSITY OF MARYLAND; CONSULTANT TO THE
UNIVERSITY HOSPITAL AND DIRECTOR OF THE CLINICAL LABORATORY;
AUTHOR OF A TREATISE ON "DISEASES OF THE STOMACH," ETC.

IN TWO VOLUMES

VOLUME I

ANATOMY, PHYSIOLOGY, INTESTINAL BACTERIA, METHODS OF DIAGNOSIS,
THERAPY AND MATERIA MEDICA OF INTESTINAL DISEASES, DIARRHEA,
CONSTIPATION, ENTERALGIA AND ENTERODYNIA, METEORISM,
DYSTRYPSIA, ENTERITIS, COLITIS, DYSENTERY,
INTESTINAL ULCERS, INTESTINAL
NEOPLASMS, ETC.

With many Original Illustrations, some of which are in Colors

LANE MEDICAL LIBRARY
4643
SAN FRANCISCO

PHILADELPHIA
P. BLAKISTON'S SON & CO.
1012 WALNUT STREET
1901

COPYRIGHT, 1901,
BY P. BLAKISTON'S SON & CO.

WM. F. FELL & CO.,
ELECTROTYPERS AND PRINTERS,
1220-24 SANSOM STREET,
PHILADELPHIA.

L060
H489
1901-2

TO

Reginald H. Fitz, LL.D., M.D.,

HERSEY PROFESSOR OF THE THEORY AND PRACTICE OF PHYSIC,
HARVARD UNIVERSITY, BOSTON,

THIS VOLUME IS RESPECTFULLY DEDICATED
IN GRATEFUL ACKNOWLEDGMENT OF HIS MANY SCHOLARLY CONTRIBUTIONS
MADE TO MEDICAL SCIENCE,
ESPECIALLY TO THE PATHOLOGY AND THERAPY OF INTESTINAL DISEASES
WHICH, IN THE LANGUAGE OF TALAMON, WERE "EPOCH MAKING."

August, 1901. THE AUTHOR.

PREFACE.

When this work was begun, there was no complete treatise on diseases of the intestines of American origin.

Aside from the fact that the German and French works on this subject are intelligible to but very few English-speaking practitioners, our climate, habits of life, and the nature of our diet and consequently the etiology of many intestinal diseases are sufficiently distinctive to merit study and elucidation in a special work. A number of entirely original methods of diagnosis, chemic researches, and histologic studies of certain intestinal diseases are here fully set forth for the first time, and an effort has been made to do justice to the work of American and English clinicians in this special department.

In this connection I consider it my duty to emphasize that two of the most fundamental and masterly works in the history of medicine on diseases of the digestive tract have been written by Americans. I refer to the researches and clinical studies of Beaumont on the Stomach, of Alexis St. Martin, and the monumental work of J. J. Woodward on (Diarrhea, Dysentery, Tuberculosis) "Diseases of the Intestines" published by the United States Government in that magnificently illustrated volume—the "Medical Report of the War of the Rebellion." These contributions have been far reaching in their educational and scientific influence and have commanded the respect and admiration of foreign clinicians. If I claim, therefore, a certain historic familiarity and the credit of pioneer work for American clinicians it will find its justification in these masterly works.

My own efforts in this present publication have been directed to furnishing the practitioner with a complete work from which he may readily instruct himself concerning the most approved and modern methods of diagnosis and treatment of intestinal diseases.

As can be seen from the bibliography quoted, almost the entire modern literature on this subject has been gone over and systematized, and, after the application of conservative critical judgment, I have attempted to derive from this literature those scientific deductions that appeared of genuine value, marked a distinct advance, or promised an actual help to diagnosis and treatment.

Concerning the tendencies of specialization in American medicine, I refer to what has been said in the preface to the first and second editions of my work on "Diseases of the Stomach." The same facts hold good with regard to a special work on "Diseases of the Intestines."

Three chapters of this work have been written by associate professors in the University of Maryland.

The section on the "Anatomy and Histology of the Intestines" is from the pen of J. Holmes Smith, M.D., Associate Professor of Anatomy, University of Maryland.

The section on the "Examination of the Feces and Urine" is by Harry Alder, M.D., Associate Professor of Diseases of the Stomach and Demonstrator in Clinical Pathology in the University of Maryland.

The section on the "Intestinal Bacteria" was written by Wm. Royal Stokes, M.D., Associate Professor of Pathology and Bacteriology, University of Maryland, and Bacteriologist in the Department of Public Health of Baltimore.

The section on "Diseases of the Rectum" is from the pen of Dr. Thomas Charles Martin, Professor of Proctology, Cleveland College of Physicians and Surgeons.

It gives me pleasure to express my thanks to these gentlemen for their scholarly contributions on their various subjects.

I am under obligations to Prof. William H. Welch for loaning and presenting a number of specimens of pathologic conditions of the intestines. A large number of the photographs for the plates were in part obtained through his kindness from the Pathologic Museum of the Johns Hopkins Hospital. The histological plates and anatomical illustrations are from the Anatomical Laboratory of the University of Maryland, with three exceptions, which were kindly loaned by Prof. Henry M. Hurd from articles published by Prof. F. P. Mall in the "Hopkins Hospital Bulletin." Three plates in the article on "Appendicitis" have been loaned by having originally appeared in Deaver's "Treatise on second edition. Six anatomic illustrations of rare e with Dr. Deaver's kind permission, been inserted fro "Surgical Anatomy." The remaining drawings were mac artists, Miss Cleveland and Mr. Louis Schmidt.

An important part of the creation of this work has fallen t lot of my associate, Dr. Harry Adler. I refer to the reading a criticism of the proof. He has brought to this laborious t mature experience gained in r[illegible]g the entire proof of the se edition of my work on "Diseases of the Stomach," and it gives pleasure to make grateful acknowledgment of his diligence. indexes of authors and of subjects have been compiled l assistants, Dr. Wilbur F. Skillman and Dr. Charles C. Conse

A critical inspection of the typographic and illustrative p the work and its general style can not fail to impress the r reader with the thoroughness of the work of my publishers, P. Blakiston's Son & Co.; work that gives evidence of a very conception and of the ability of their publishing department.

To the Medical Faculty of the University of Maryland grateful for many kind acts in supporting the clinical laborat and making this work possible by the appointment of willi assistants.

J. C. H.

August, 1901.

TABLE OF CONTENTS.—VOL. I.

PART FIRST.

ANATOMY, HISTOLOGY, PHYSIOLOGY, BACTERIOLOGY, AND PATHOLOGY OF THE INTESTINES.—PHYSICAL METHODS OF DIAGNOSIS.

CHAPTER I.

PAGE

ANATOMY AND HISTOLOGY OF THE INTESTINES.—ANATOMICAL ASPECT OF THE ABNORMALITIES OF POSITION AND FORM, ... 17–35

The Small Intestine.—The Large Intestine.

CHAPTER II.

PHYSIOLOGY OF THE INTESTINES, ... 36–61

The Alkaline Succus Entericus.—The Pancreatic Juice.—The Bile.—The Relation of Gastric to Duodenal Secretion. Preparatory Secretion in the Duodenum.—The Effect of Gastric Anacidity and Hyperacidity upon Duodenal Digestion.—The Specific Irritability of the Various Portions of the Gastro-intestinal Canal and the Special Food Excitants to the Digestive Secretions.

CHAPTER III.

ABSORPTION FROM THE INTESTINES, ... 62–83

Paths of Absorption.—The Absorption of Water.—The Absorption of Salts and Soluble Constituents by the Columnar Cells Lining the Intestines.—The Absorption of Carbohydrates.—Conditions of Absorption of Proteids.—Changes in Albumose and Peptone Occurring During Absorption.—The Digestion and Absorption of Fats.—Literature.

CHAPTER IV.

THE PERISTALSIS OF THE INTESTINES, ... 84–104

Innervation of the Intestines.—Influence of Disturbances of Circulation on the Peristalsis.—Pathological Intestinal Peristalsis.—Defecation.—The Influence of Medicinal Agents on the Peristalsis.

CHAPTER V. PAGE

UTILIZATION OF FOOD IN THE STOMACH AND INTESTINES OF THE HEALTHY MAN AND UNDER ABNORMAL CONDITIONS, 105–123

Kinds of Food.—Composition, Amount, and Preparation of Food.—Behavior of the Healthy Intestine During Digestion.—Utilization of Food in the Intestines under Abnormal Conditions.—Digestibility of the Various Foods.—Proteolysis at First Rapid, May be Arrested Later by its Own Products.—Importance of Spices and Relishes in Nutrition and Digestion.—Results of Hypernutrition.

CHAPTER VI.

DISTURBANCES IN THE SECRETORY FUNCTIONS OF THE INTESTINES, THE PANCREAS, AND THE LIVER, ... 124–134

Disturbances in the Secretion of the Intestinal Juice (Succus Entericus).—Detrimental Results of Decrease in the Secretion of the Intestinal Juice for the Digestion and Absorption of Foods.—Disturbances in the Formation of Bile and in its Flow into the Intestines.—Injurious Results of an Obstructed Flow of the Bile into the Intestines, for the Digestion and Absorption of Foods.—Disturbances in the Secretion of the Pancreatic Juice.—Results of the Absence of the Pancreatic Juice for the Digestion of Food Substances.

CHAPTER VII.

THE INTESTINAL BACTERIA, ... 135–204

Bacillus Coli Communis (Escherich).—Bacillus Lactis Aërogenes (Escherich).—Fermentation Formula of the Colon Bacillus.—The Intermediate, or Hog Cholera, Group.—Chief Distinguishing Features of the Eberth, or Typhoid, Gärtner, or Intermediate, and Escherich, or Colon, Groups.—Bacillus Subtilis (Ehrenberg).—Bacillus Butyricus (Prazmowski).—Bacillus Fecalis Alcaligenes (Petruschky).—Bacillus Putrificus Coli (Bienstock).—Bacillus Aërogenes (Welch).—Imperfectly Described Forms.—The Micrococci of the Intestines.—Blastomycetes, or Yeasts.—Difficulties of Study.—The Bacteria which are Found in the Various Diseases of the Intestines.—The Spirillum of Asiatic Cholera (Koch).—The Pathogenic Protozoa of the Intestines.—The Influence which Bacteria Exert on the Processes of Digestion.—Literature.

CHAPTER VIII.

METHODS AND TECHNICS OF DIAGNOSIS, ... 205–228

Methods of Examination.

CHAPTER IX.

DIAGNOSTIC SIGNIFICANCE OF EXAMINATION OF FECES AND URINE, ... 229–258

Feces.—Urine.—Literature.

CHAPTER X.

DIAGNOSTIC SIGNIFICANCE OF CHEMICAL AND PHYSICAL EXAMINATION OF THE GASTRIC FUNCTIONS FOR INTESTINAL DISEASES, ... 259–262

CHAPTER XI.

DUODENAL INTUBATION, ... 263–272

PART SECOND.

DIET.—THERAPY AND MATERIA MEDICA OF INTESTINAL DISEASES.

CHAPTER XII. PAGE

PRINCIPLES OF DIETETIC TREATMENT.—THE DIETARY PROPHYLAXIS AND HYGIENE.—SPECIAL DIETARY PROPHYLAXIS.—EXTRABUCCAL FEEDING. RECTAL AND SUBCUTANEOUS ALIMENTATION, ... 273–315

Diet During Epidemics of Cholera, Typhoid Fever, and Dysentery.—Intestinal Indigestion (Dystrypsia).—Habitual Constipation. Hemorrhoids.—Severe Acute Intestinal Catarrh (Diarrhea), Diarrhea with Vomiting, Cholera, Acute Dysentery, Acute Inflammation of the Appendix.—Mild Intestinal Catarrh (Diarrhea), Chronic Dysentery, Intestinal Tuberculosis, Convalescence, Inflammation of the Cecum.—Physiology of Rectal Digestion.—Preparation of Rectal Enemata.

CHAPTER XIII.

METHODS AND MEANS OF TREATMENT, ... 316–349

Mineral Springs.—The More Important Medicinal Agents.—Intestinal Autointoxication—Intestinal Antiseptics.—Massage.—Hydriatic Treatment of Intestinal Diseases.—Treatment by Electricity.

PART THIRD.

INTESTINAL CLINIC.

CHAPTER XIV.

DIARRHEA, ... 350–355

Nature and Concept.—Etiology.—Dyspeptic and Dystryptic Diarrhea.—Cathartic Diarrhea.—Stercoral Diarrhea.—Entozoic Diarrhea.—Nervous Diarrhea.—Diarrhea After Taking Cold.—Diarrhea Caused by Irritants Acting from the Blood.—Treatment.—The Diet.

CHAPTER XV.

OBSTIPATION OR CONSTIPATION—COPROSTASIS, ... 356–393

Concept and Nature of Constipation.—Etiology.—Chronic Constipation from Disease.—Résumé of Causes Coming to the Foreground in Modern Life.—Symptomatology.—Diagnosis.—Prognosis.—Treatment.

CHAPTER XVI.

INTESTINAL COLIC—ENTERODYNIA, ... 394–396

Etiology.—Symptomatology.—Diagnosis.—Treatment.

CHAPTER XVII. PAGE

METEORISM—TYMPANITES, ... 397–404

Variety of Intestinal Gases.—Etiology of Meteorism.—Symptomatology and Diagnosis.—Treatment.

CHAPTER XVIII.

INTESTINAL INDIGESTION—DYSTRYPSIA INTESTINALIS, . . . 405–432

Nature and Concept.—I. Intestinal Indigestion or Dystrypsia Due to Demonstrable Pathologico-anatomical Alterations in the Structure of the Intestinal Walls, Lymphatics, Blood-vessels, and Nerves.—II. Absence or Deficiency of Intestinal Digestive Secretions, Principally of Bile and Pancreatic Juice.—III. Intestinal Indigestion Due to Qualitative or Quantitative Irregularities of the Diet.—IV. Intestinal Indigestion Due to Abnormal Bacterial Activity.—V. Intestinal Dystrypsia Due to Abnormal Gastric Chemistry.—VI. Intestinal Dystrypsia of Nervous Origin (Neurasthenia Intestinalis).—VII. Intestinal Indigestion Caused by Abnormal Substances or Irritants Reaching the Intestine from the Blood.—VIII. Dystrypsia Due to the Activity of Intestinal Parasites—Worms.—IX. Dystrypsia Due to Hyper- or Hypoperistalsis.—General Considerations Concerning Intestinal Dystrypsia.—Treatment.

CHAPTER XIX.

ENTERITIS (INTESTINAL CATARRH), ... 433–514

1. Acute Enteritis (Acute Intestinal Catarrh; Enteritis Acuta).—2. Chronic Enteritis.—3. Phlegmonous Enteritis (Purulent Inflammation of the Intestines).—4. Diphtheric, Croupous, or Pseudomembranous Enteritis—Diphtheric Dysentery or Ulcerative Colitis.—5. Membranous Colitis or Enteritis and Mucous Colic.—Sigmoiditis and Pericolitis.—Literature.

CHAPTER XX.

DYSENTERY, ... 515–591

Acute and Chronic Dysentery.

CHAPTER XXI.

ULCERS OF THE INTESTINE, ... 592–669

History.—Ulceration in Consequence of Necrobiotic Processes.—Ulceration Due to Inflammatory Processes.—Ulcerations Due to Acute Infectious Diseases.—Ulcerations Occurring in Chronic Infectious Diseases.—Ulcerations Occurring in Constitutional Diseases—Gout.—Toxic Ulcerations.—Symptomatology and Diagnosis.—Prognosis.—Treatment.—Literature.

CHAPTER XXII.

INTESTINAL NEOPLASMS, ... 670–730

I. Malignant Neoplasms (Carcinoma, Sarcoma, and Lymphosarcoma).—Sarcoma and Lymphosarcoma of the Intestine.—Benign Neoplasms of the Intestines.—Literature.

INDEX OF AUTHORS, ... 731

INDEX OF SUBJECTS, ... 737

LIST OF ILLUSTRATIONS.

PLATE PAGE
I. Abdominal Viscera; Great Omentum. (From Deaver's "Surgical Anatomy.") . . . . *Facing Page* 18
II. Right Side of Mesentery, Cecum, Ascending and Transverse Colon. (From Deaver's "Surgical Anatomy.") . . . . *Facing Page* 20
III. Fig. 1. General View of the Vascular Branches Going in and out of Heller's Plexus, ... *Facing Page* 22
Fig. 2. General View of the Course and Distribution of the Blood-vessels in the Entire Intestine, ... *Facing Page* 22
IV. Fig. 1. Diagrammatic Representation of the Coats of the Lymph-vessels in the Intestinal Mucosa, ... *Facing Page* 28
Fig. 2. General View of the Lymph-vessels in the Intestinal Walls, *Facing Page* 28
V. Pelvic Cavity of Male (Superior View). (From Deaver's "Surgical Anatomy.") . . . . *Facing Page* 30
VI. Ileocecal Valve and Cecum, ... *Facing Page* 34

FIG. PAGE
1. Relation of Portal Vein and Superior Mesenteric Artery to the Duodenum and Pancreas, ... 23
2. Usual Position of the Intestine in the Abdominal Cavity, ... 25
3. Ileocecal Valve, Opening of Appendix, ... 29
4. Ascending and Descending Colon, Sigmoid Flexure, ... 32
5. Interior of Invaginated Ileum, ... 33
6. Plaster Casts of Duodenum of Infant and Adult, ... 55
7. Gas-production in Sugar-free Bouillon Containing 1 Per Cent. Dextrose. The Daily Production of Gas is Indicated by the Short Transverse Lines. Intermediate and Colon Group, ... 149
8. Gas-production of Intermediate Group in Sugar-free Bouillon Containing 1 Per Cent. Dextrose, ... 151
9. Typical Reactions of Typhoid, Intermediate, and Colon Group in a 1 Per Cent. Lactose Sugar-free Bouillon, ... 153
10. Reaction of Typhoid, Intermediate, and Colon Group in 1 Per Cent. Saccharose. Bubble at Top Due to Slight Conversion of Saccharose During Sterilization, ... 155
11. Reactions in Sugar-free Bouillon Containing 1 Per Cent. Saccharose. Typhoid, Intermediate, and Colon Group, ... 157
12. A Positive Typhoid Reaction. Typhoid Bacilli in a Bouillon Culture with which Typhoid Diluted Blood-serum has been Mixed, ... 175
13. A Negative Typhoid Reaction. Typhoid Bacilli in a Bouillon Culture, as seen under a High Magnifying Power, Before the Addition of Typhoid Blood-serum, ... 175

FIG. PAGE
14. Boas' Stool-sieve, ... 230
15. Apparatus for Obtaining Intestinal Contents, ... 264
16. Pressure Bottles for Distending the Intragastric Bag During Duodenal Intubation, ... 265
17. Duodenal Tube, ... 269
18. Balloon Sound of Kuhn, ... 270
19. Esophageal Sound, ... 270
20. Action of an Ordinary Stomach-tube on Attempting to Push through Pylorus, . 271
21. The Kuhn Duodenal Tube Passing through the Stomach, ... 271
22. Amœba Coli, ... 521
23. Amebic Dysentery (Colon), ... 529
24. Histology of Amebic Dysentery, Section from Cecum, ... 531
25. Healing Dysenteric Ulceration of Colon, ... 557
26. Diphtheric Dysentery (Colon), ... 563
27. Chronic Dysenteric Ulceration of Colon, ... 567
28. Dysenteric Ulceration of Colon, ... 571
29. Diphtheric and Hemorrhagic Colitis, ... 575
30. Diphtheric Colitis, ... 579
31. Tuberculous Ulceration of Ileum, ... 633
32. Tuberculous Ulceration of Ileum, ... 635
33. Tuberculous Ulceration of Ileum, ... 639
34. Tuberculous Ulceration of Sigmoid Flexure, ... 641
35. Histology of Tubercular Ulcer, ... 645
36. Adenocarcinoma of Sigmoid Flexure, ... 671
37. Adenocarcinoma of Colon with Pseudomembranous Colitis, ... 675
38. Lymphosarcomatous Nodules in Intestine, ... 699
39. Secondary Sarcoma of Ileum, ... 703
40. Multiple Polypi of Small Intestine, ... 709
41. Mucous Polypi of Colon, ... 711
42. Polypoid Colitis (Colitis Polyposa), ... 713
43. Mucous Polypi of Colon, ... 717
44. Submucous Lipoma of Colon, ... 719
45. Intestinal Polypi, ... 721

DISEASES OF THE INTESTINES.

PART FIRST.

ANATOMY, HISTOLOGY, PHYSIOLOGY, BACTERIOLOGY, AND PATHOLOGY OF THE INTESTINES.—PHYSICAL METHODS OF DIAGNOSIS.

CHAPTER I.

ANATOMY AND HISTOLOGY OF THE INTESTINES.—ANATOMICAL ASPECT OF THE ABNORMALITIES OF POSITION AND FORM.

By J. HOLMES SMITH, M.D.,

ASSOCIATE PROFESSOR OF ANATOMY, UNIVERSITY OF MARYLAND.

The intestinal tract consists of two portions—the small and the large intestine. The small intestine begins at the pylorus and terminates at the ileocecal valve. The large intestine consists of two portions—the colon and the rectum. This chapter will be devoted to a special consideration of the small intestine and the colon, the latter beginning at the cecum and terminating in the rectum.

THE SMALL INTESTINE.

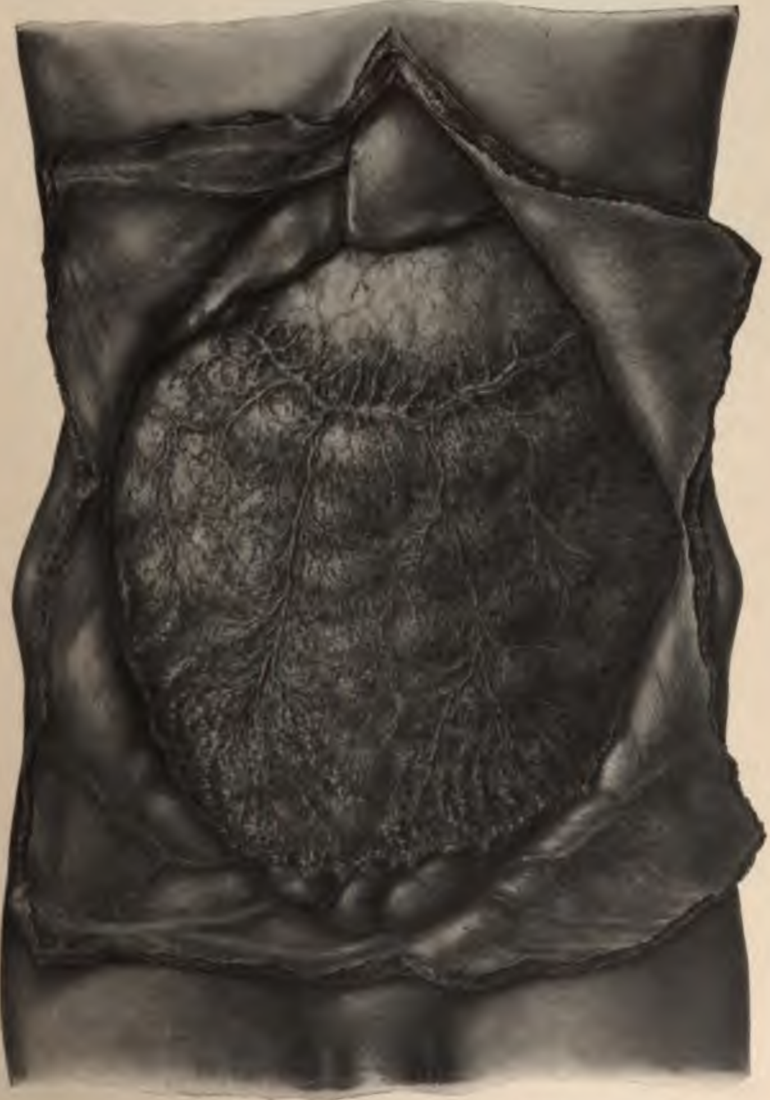
The small intestine is a muscular membranous tube, and is divided into three parts—duodenum, jejunum, and ileum. It is arranged in a series of coils, and is contained mostly in the umbilical, lumbar, and hypogastric regions, with quite a considerable portion of the ileum in the pelvis, although coils of small intestine may be found in any region of the abdominal cavity. The average length is about twenty feet, but this is subject to great variations, Treves giving the extreme limit of the whole intestinal tract as from fifteen feet six inches to thirty-one feet ten inches. In one case measured in the anatomical laboratory of the University of Maryland the small intestine alone measured thirty-one feet and six inches. The length of the intestinal canal does not seem to bear a fixed relation to the size and weight of the individual, and there are clinical reasons for believing that the development of the intestinal tract may be influenced by certain diseases occurring in childhood —particularly enterocolitis.

The lumen of the small intestine is larger at its upper end, and gradually grows smaller as it goes downward, but the diminution is so gradual, considering the length of the canal, that it does not justify its being described as funnel-shaped. In structure the small intestine consists of four coats—the external serous, or peritoneal, coat (serosa), which forms an almost complete investment for the whole length of the small intestine excepting the duodenum. Beneath this coat is the muscular coat (the muscularis), which is arranged in two distinct layers—an outer longitudinal and an inner circular. The longitudinal fibers are distributed pretty evenly over the whole surface of the intestine, being a little more distinct along its free border. The circular fibers, as their name implies, surround the intestine, the contraction of the circular fibers narrowing the lumen of the intestine, producing a vermicular movement, or peristalsis, with the direction from above downward, in this way forcing along the contents of the tube. Beneath or internal to the circular or muscular coat is a layer of fibrous tissue, the submucosa, which is intimately attached to the inner coat of the intestine, and so arranged as to allow of dilation of the canal without traction upon the mucous coat.

The mucous membrane (*mucosa*) *of the small intestine* consists of a layer of plain muscular tissue, the *muscularis mucosæ*, which supports a layer of retiform tissue, which in turn supports the blood-vessels, nerves, and lymphatics of the mucous membrane. This in turn is covered by a delicate layer of endothelial cells which support the columnar epithelium lining the intestine. The mucous membrane of the small intestine is thrown into a number of folds, called the valvulæ conniventes, which project into the lumen; these folds are about one-third of an inch in depth, and, as a rule, extend about

PLATE I.

ant. omentum



ABDOMINAL VISCERA; GREAT OMENTUM.
Liver and stomach in normal position.—(*From Deaver's "Surgical Anatomy."*)

two-thirds of the way around, with an occasional fold extending all the way around the intestines. They terminate usually by dividing into two projections, these sometimes reuniting and inclosing a small free space. The valvulæ conniventes serve a double purpose —affording a much larger surface for absorption and retarding the passage of the contents of the canal. They are larger in the upper part of the intestine, beginning a short distance below the pylorus, and gradually becoming smaller, until they completely disappear at about the junction of the jejunum and ileum, the largest ones being around the point where the common bile-duct and pancreatic duct open into the duodenum.

The whole surface of the mucous membrane of the small intestine is covered with minute projections which give it a velvety appearance; these projections are the villi; they are larger and more numerous in the duodenum and jejunum, and smaller and fewer in number in the ileum; they also differ in shape and size in the different portions of the intestinal tract, those in the duodenum and jejunum being shorter and club-shaped, and in the ileum slender or filiform; in length they measure about 0.5 mm.

Each *villus* consists of a projection of retiform lymphoid tissue, supporting blood-vessels, nerves, and lymphatics, and a few fibers of muscular tissue from the muscularis mucosæ, and covered over by a delicate layer of flattened cells which forms a basement membrane upon which rest the columnar epithelial cells, the cells of this basement membrane being connected with the prolongation from the retiform tissue beneath, and sending delicate filaments between the epithelial cells above. The lacteal occupies the center of the villus, and in man is usually single and club-shaped, but occasionally double, a condition which is normal in some of the lower animals. The lacteal is surrounded by a delicate layer of endothelial cells which connect with the retiform tissue, and this in turn with the basement membrane of the villus, from which, as already stated, delicate filaments project between the epithelial cells.

The muscular tissue of the villus, which is unstriped, is disposed longitudinally around the lacteal, and is derived, as has already been stated, from the muscularis mucosæ, and arranged longitudinally around the lacteal. The epithelium covering the villus is distinctly columnar, and in the lower part of the intestinal canal a number of leukocytes are to be seen between the cells, especially in the neighborhood of the lymphoid tissue of the lower bowel. A number of mucous cells in various stages of development will also be found between the columnar epithelial cells of the villus.

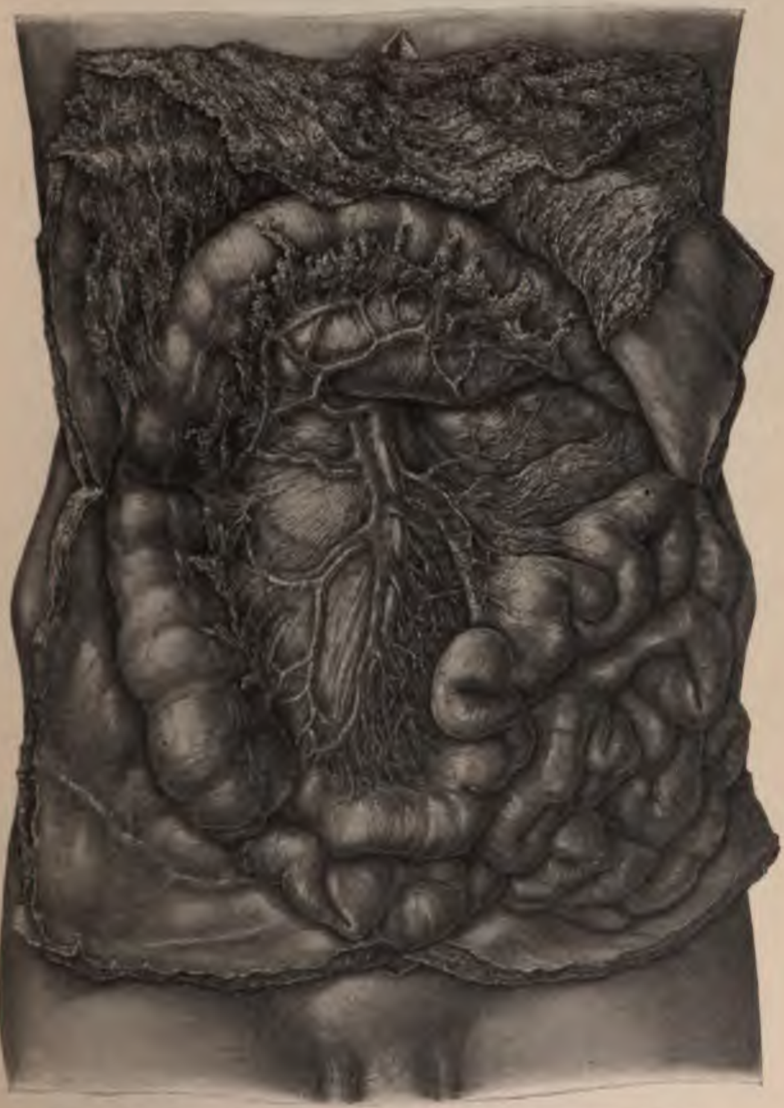
The nerves of the villi are derived from Meissner's plexus of the sympathetic, and ramify through the trabecular tissue. There is usually but one arterial branch to a villus, which passes up near the center to near its apex, where it breaks up into a capillary network, from which the blood is returned by small veins which pass out at the base of the villi to join the venous plexus in the submucosa.

The *glands of the small intestine* consist of the follicles of Lieberkühn, or simple follicles, the glands of Brunner, or duodenal glands, the solitary glands, and the agminated glands, or Peyer's patches.

The simple follicles are merely inversions of the mucous membrane projecting downward into the submucosa, and lined with the columnar epithelium, similar to that lining the remainder of the canal, between the cells of which are found a certain number of goblet cells. The epithelial cells rest upon a delicate basement membrane. Surrounding the follicle is a fine network of capillary vessels. These follicles open upon the free surface of the intestine, and their openings can be seen with a magnifying glass, appearing as minute drops between the villi. They are found throughout the whole length of the small intestine.

The duodenal glands, or glands of Brunner, are small adenoid bodies in the submucosa and opening on the free surface of the mucous membrane through a delicate duct. These glands are limited to the duodenum, and belong to the compound racemose variety.

The solitary glands differ only in arrangement and distribution, being similar in structure. The solitary or agminated glands, or Peyer's patches, are true lymphoid tissue, having no openings on the free surface of the intestine; they are situated in the submucosa, and belong to the lymphatic system, being surrounded by a plexus of lymphatic vessels, with which they communicate; this arrangement is well seen in the accompanying illustration from F. P. Mall's "Blut- u. Lymphwege im Dünndarm des Hundes," Leipzig, 1887. The solitary glands are found throughout the whole length of the



RIGHT SIDE OF MESENTERY, CECUM, ASCENDING AND TRANSVERSE COLON.
The great omentum is drawn up to show the transverse colon and mesenteric vessels.—(*From Deaver's "Surgical Anatomy."*)

small intestine, while Peyer's patches are more numerous and larger in the lower part of the ileum, but may be found even in the jejunum; they are oblong or quadrilateral in outline, and consist of a number of lymph follicles grouped and held together by adenoid tissue; they are always found at that part of the intestine which is furthest from the mesenteric attachment—that is, the distal border of the gut. They are seen as projections pushing up the mucous membrane, and are covered with villi, excepting at the apical points.

The *blood supply of the small intestine*, excepting part of the duodenum, is derived from the superior mesenteric artery, which comes off from the abdominal aorta just below the celiac axis, and passes downward and toward the right in a curved line, with the convexity to the right.

From the convex side large branches are given off,—vasa intestini tenuis,—which pass forward between the layers of the mesentery and break up into a series of arterial arches—from the distal ones, of which the straight vessels are given off which go to the proximal border of the intestine and send branches around it, anastomosing along the free side of the intestine, that is, opposite to the insertion of the mesentery, the lateral anastomosis between adjacent branches being slight. Branches of these vessels pierce the muscular coat, supplying it, and, ramifying through the submucosa, form plexuses around the intestinal glands and send small branches to the villi, the distribution of which has already been given in the description of the villi. The duodenum is supplied by the pancreaticoduodenalis superior from the gastroduodenal, and the pancreaticoduodenalis inferior from the superior mesenteric.

The *veins of the small intestine* form large plexuses in the submucosa, which collect blood from the villi and glands, and piercing the muscular coat, form the radicles of the superior mesenteric vein, which, passing upward alongside of the artery, helps to form the portal vein. The arrangement and distribution of the arteries and veins are schematically illustrated in accompanying plate, from Mall's article (*l. c.*).

The *nerves* arise from the solar plexus, and follow the branches of the artery to the proximal border of intestine; piercing the outer layers of the muscular coat they form a series of plexuses (Auerbach's), giving off branches which pierce the circular muscular layer and form a second series of plexuses in the submucosa (Meissner's), from which in turn the filaments which supply the mucosa originate.

The *lacteals* lying in the villi pass to the lymphatic plexuses in the submucosa; from here branches pierce the circular muscular coat and form a second series of plexuses, from which other branches pierce the longitudinal muscular layer and pass between the mesenteric layers to the receptacle of the chyle, communicating freely with the mesenteric glands.

The *lymphatics* of the submucosa form a series of plexuses which surround the glands, especially the solitary and Peyer's patches, and communicate freely with the lymph-spaces of those bodies.

The Duodenum.

The duodenum is the first part of the small intestine, and is from ten to twelve inches long, curved around the head of the pancreas, and divided into three portions. The first, or ascending, part is about two inches long and passes upward, backward, and outward to the under surface of the liver; the second, or descending, part passes downward in front of the right kidney, to which it is attached, as far as the third lumbar vertebra; the third, or transverse, portion crosses the vertebral column, passing slightly upward to terminate in the jejunum. Its caliber is larger and its coats are thicker than the other portions of the small intestine.

The first part is the most movable, and has a peritoneal investment similar to that of the stomach.

The second part closely encircles the head of the pancreas, to which it is firmly attached. This part receives the pancreatic and common bile-ducts.

The third portion, passing across the vertebral column in front of the vena cava and aorta, is fixed by the superior mesenteric artery and portal vein (see illustration of relation of duodenum to pancreas and superior mesenteric artery).

The terminal portion of the duodenum, sometimes described as the fourth part, projects to the left of the mesenteric vessels, and is held by a ligament containing some muscular tissue derived from the left crus of the diaphragm; this part has a peritoneal investment similar to that of the jejunum.

The second and third portions of the duodenum are the most

PLATE III.

From F. P. Mall, "Die Blut- und Lymphwege im Dünndarm des Hundes." (Des XIV. Bandes der "Abhandlungen der mathematisch-physischen Classe der Königl. Sächsischen Gesellschaft der Wissenschaften.")

FIG. 1.—General View of the Vascular Branches Going in and out of Heller's Plexus. Arteries, red; capillaries, colorless—outlined in black lines; veins, blue.

FIG. 2.—General View of the Course and Distribution of the Blood-vessels in the Entire Intestine, imitating, as far as possible, actual conditions as they are found in the very much distended intestine. The venous dilations are not included. The mucosa is white, the muscularis mucosæ, dark yellow; the submucosa, gray; the circular muscular fiber, pink; the longitudinal muscular fiber, light yellow. The arteries are cinnabar red; the veins, Berlin blue. Where the mucosa has been cut out at *s*, *s'*, and *s''*, the course and distribution of the shorter vessels are visible in the submucosa. These are on the inner side of the submucosa. The outer side of the submucosa is visible where the muscular layer has been cut away, and here we see the course and distribution of the longer and recurrent vessels. The artery is drawn in as being external to the vein, which is the reverse of the true anatomical relation, but has been done for the

PLATE III.

Fig. 2.

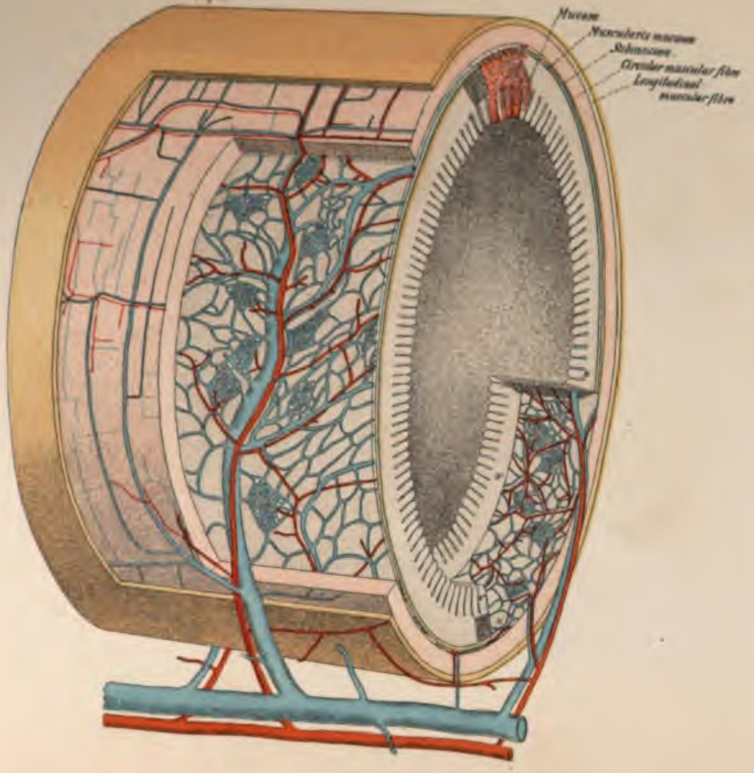
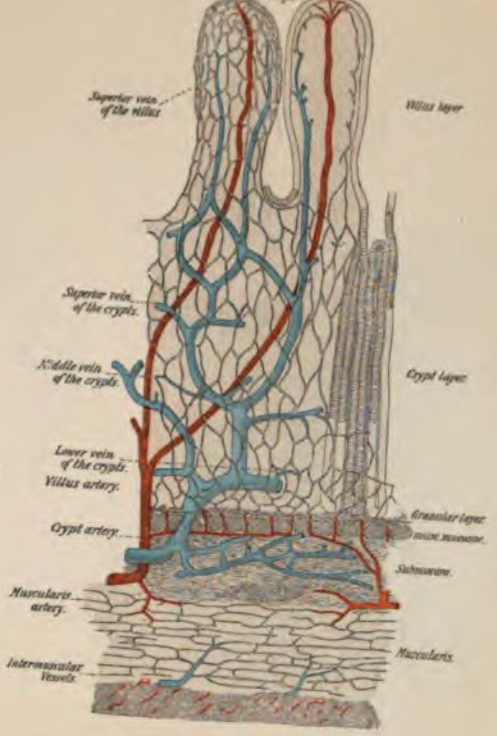


Fig. 1



fixed portions of the intestine; this fixation renders the upper part of the intestine liable to rupture from blows upon the abdomen when the intestine is distended; also to compression from neoplasms, floating kidney, etc., because it can not escape the compressing body, owing to its fixation.

Owing to the relation of the first portions of the duodenum to the gall-bladder and colon, it is easy to understand how adhesions and subsequent fistulous openings may occur, allowing the passage of gall-stones into the upper part or fistulous openings between the colon and the second part.

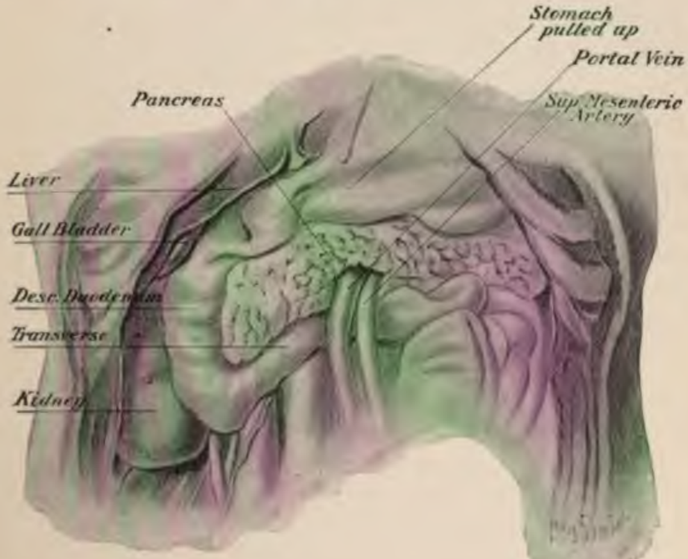


FIG. 1.—RELATION OF PORTAL VEIN AND SUPERIOR MESENTERIC ARTERY TO THE DUODENUM AND PANCREAS.—(*From dissection in the anatomical laboratory of the University of Maryland.*)

The duodenum may be obstructed by tumors or cysts.

In a case in the wards of the hospital of the University of Maryland in 1899, partial obstruction of the duodenum causing repeated attacks of vomiting of large quantities of bile, the obstruction was caused by enlargement of mesenteric glands near the root of the superior mesenteric artery; this case was permanently relieved by operation.

Allen * mentions two cases of aneurysm of the superior mesenteric artery reported by J. A. Wilson † in which pressure of the aneurysmal sac had interfered with the flow of bile and induced jaundice, and one by Gairdner, ‡ in which an aneurysm of the superior mesenteric artery had burst into the duodenum, causing repeated attacks of hematemesis.

The Jejunum and the Ileum.

The jejunum and the ileum together comprise nearly the whole length of the small intestine, for they extend from the termination of the duodenum at the left side of the second lumbar vertebra, and terminate at the ileocecal valve. The general direction, corresponding to that of the direction of the mesentery, is from above downward and to the right. While the division of this portion of the intestine into two parts is rather arbitrary, yet there are differences which serve to distinguish the upper from the lower part of the canal. The jejunum makes up about the upper two-fifths, and all its coats are thicker than those of the ileum, and its mucous membrane is arranged in valvulæ conniventes, which disappear in the ileum. The lower three-fifths—the ileum—is much thinner than the jejunal part. The jejunoileum is arranged in a series of coils which are suspended from the vertebral column by the layers of the peritoneum, which form the mesentery, which passes to the proximal or attached border of the intestine, and around it to the proximal border again, and back to the vertebral column, forming in this way a sling, between the two layers of which all the vessels and structures pass which go through and come from the intestines.

The mesentery, spread out, is fan-shaped, with the intestine folded along the outer edge. The coils of the small intestine are in close apposition, with the parallel mesenteric planes of adjacent coils, those of the jejunum being for the most part nearly horizontal, while the lower coils of the ileum are arranged nearly perpendicularly.

The arrangement of these coils and their position in the abdominal cavity are not matters of chance, but they bear a fixed and

---

* "Human Anatomy," vol. VI, p. 649.

† "Medico-Chir. Trans.," vol. XXIV, p. 221.

‡ "Clinical Medicine," 1862, 504.

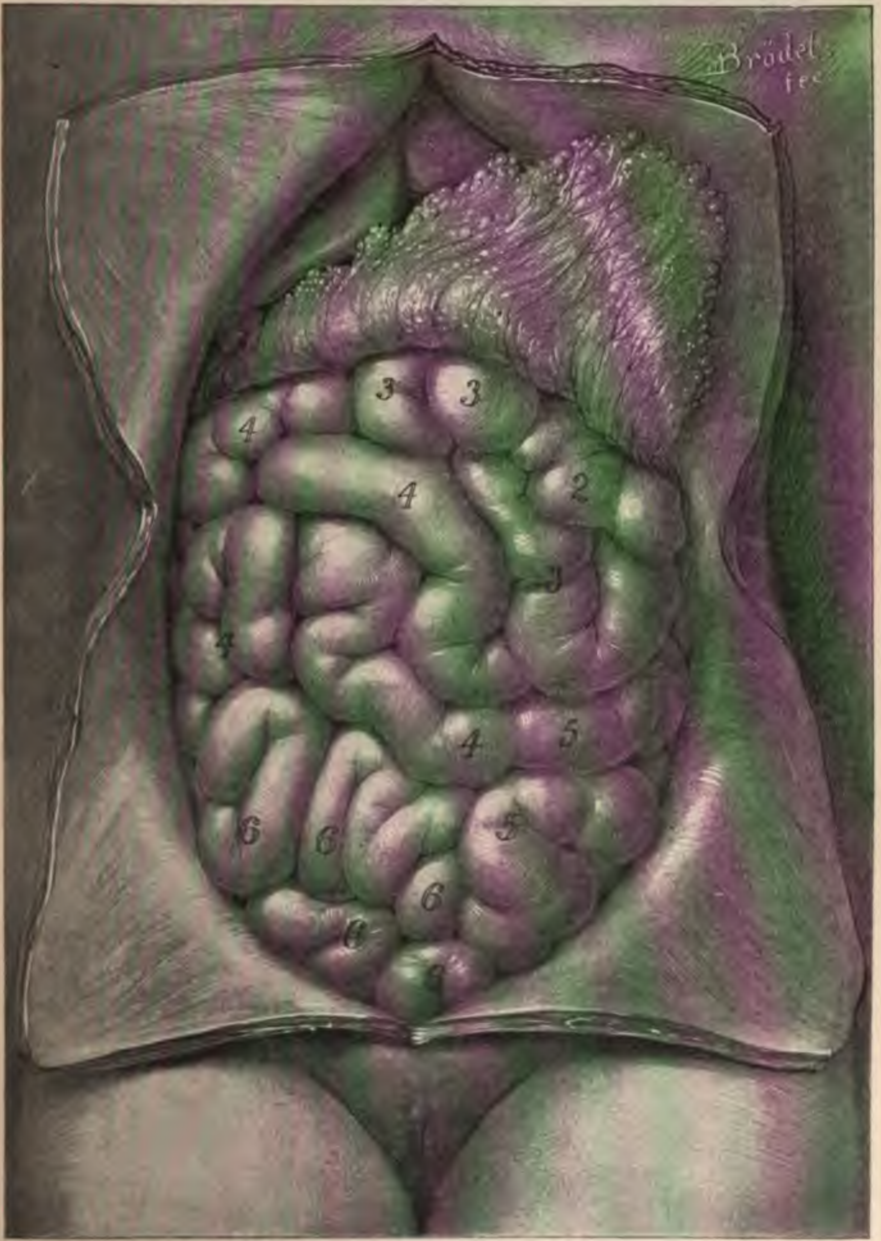


FIG. 2.—USUAL POSITION OF THE INTESTINE IN THE ABDOMINAL CAVITY.

The Arabic figures indicate the course of the intestine: 2 being nearer the duodenum and 6 nearer the cecum.

Although this is an actual specimen, it represents the condition in twenty-one out of forty-one cadavers.—(*From F. P. Mall, "Development of Human Intestine and its Position in Adult," "Johns Hopkins Hos. Bull.," No. 90 and 91, 1898.*)

definite relation to one another and to the surrounding parts, as is shown by the investigations of F. P. Mall, who has examined a great many bodies with reference to that point. These coils of intestines move freely upon one another, and, owing to the peculiarity of the mesenteric attachment, each coil has quite a wide range of movement.

The jejunum occupies principally the left lumbar and umbilical regions, and the ileum the umbilical and hypogastric districts, coils of the ileum also being found in the pelvis. The ileum terminates at the ileocecal valve, where it joins the large intestine.

The ileocecal valve is a slit-like opening in the side of the cecum, presenting two folds or lips, which are formed by the projection into the large intestine of the mucous, submucous, and circular muscular coats of the small intestine. The direction is transverse to the long axis of the large intestine. The apposing surfaces of the ileocecal valve are covered with the villi, while the surfaces which belong to the large intestine are devoid of them. This valve prevents the contents of the large intestine from passing backward into the ileum. It is sufficient against solids, excepting in case of trauma and simultaneous distention of the cecum and ileum, when it sometimes allows the passage of liquids, and always of gases, so that under these unusual conditions the whole intestinal tract may be inflated with gas through the rectum, a fact which has been taken advantage of in cases of suspected invagination (see chapter on Treatment of Invagination).

THE LARGE INTESTINE.

The large intestine consists of the cecum and vermiform appendix, the ascending, transverse, and descending colon, sigmoid colon, or sigmoid flexure of the colon, and the rectum.

The large intestine differs in structure and shape somewhat from the small intestine—instead of being smoothly cylindrical or circular it is sacculated or arranged in a series of pouches, due to the peculiar arrangement of the external longitudinal muscular coat, which, instead of being evenly distributed over the surface of the intestine, as is the case with the small intestine, is arranged in three longitudinal bands—the tæniæ coli—which are equidistant from one another—one on the anterior, one posterior, and a third one on the internal border. These muscular bands are shorter than the other coats of the intestine, and in this way the peculiar puckering or sacculation of the large intestine is produced, as is easily proved by dissecting off the tæniæ coli, or longitudinal bands, when it will be found that the large intestine will become circular when inflated. The tæniæ coli, or longitudinal bands, form an absolutely distinctive mark by which the large intestine can be distinguished from any other portion of the intestinal canal. The mucous membrane of the large intestine is smooth, and its glands consist of the simple follicles, or tubular glands, and the solitary glands, which are scattered over it, the latter being larger than those in the small intestine, but similar in structure.

The sacculation of the large intestine gives the appearance of a number of valves on looking at the interior; these projections are not to be confounded with the valvulæ conniventes of the small intestine, as the valvulæ conniventes of the small intestine are foldings of the mucous membrane alone, while the valve-like projections of the large intestine involve all its coats excepting the longitudinal muscular, and their purpose is not to afford a larger surface of absorption, but to act as a support and resistance to the too rapid passage of the intestinal contents.

The large intestine receives its *blood supply* from the superior and inferior mesenteric artery. The superior mesenteric, supplying the cecum and ascending colon through the ileocolic and colica dextra, and the transverse colon through the colica media, the descending colon being supplied by the colica sinistra of the inferior mesenterics, and the sigmoid flexure by the sigmoid branch of the same vessel. The veins of same names as arteries empty into the portal system. The vascular supply of the colon is well depicted in the accompanying illustration from Deaver's "Surgical Anatomy."

The colon derives its nerve supply from the sympathetic system.

The Cecum.

The cecum is the beginning of the large intestine; as its name implies, it is a blind pouch. It is about two and one-half to three inches in length, and extends downward from the point of entrance

PLATE IV.

From F. P. Mall, "Die Blut- und Lymphwege im Dünndarm des Hundes." (Des XIV. Bandes der "Abhandlungen der mathematisch-physischen Classe der Königl. Sächsischen Gesellschaft der Wissenschaften.")

FIG. 1.—Diagrammatic Representation of the Coats of the Lymph-vessels in the Intestinal Mucosa. Adjoining one fully extended villus there are several shorter ones showing the central cavity and the spiral tubule in the terminal end. From the network beneath the villi lymph-vessels are seen descending in a straight line, which form the lymph network of the mucosa at the base of the crypts of Lieberkühn. From there they either extend over the accumulation of the lymph-follicles or immediately into the submucosa. The muscular tunic ceases in front of the follicle.

FIG. 2.—General View of the Lymph-vessels in the Intestinal Walls. On the transverse section the mucosa is white; the muscularis mucosæ, blue; the submucosa, pink; the circular muscular fiber, yellowish red; the longitudinal muscularis, yellow. The difference in the formation of the lymphatic network in the presence or absence of lymph-follicles is noticeable.

PLATE IV.

Fig. 2.

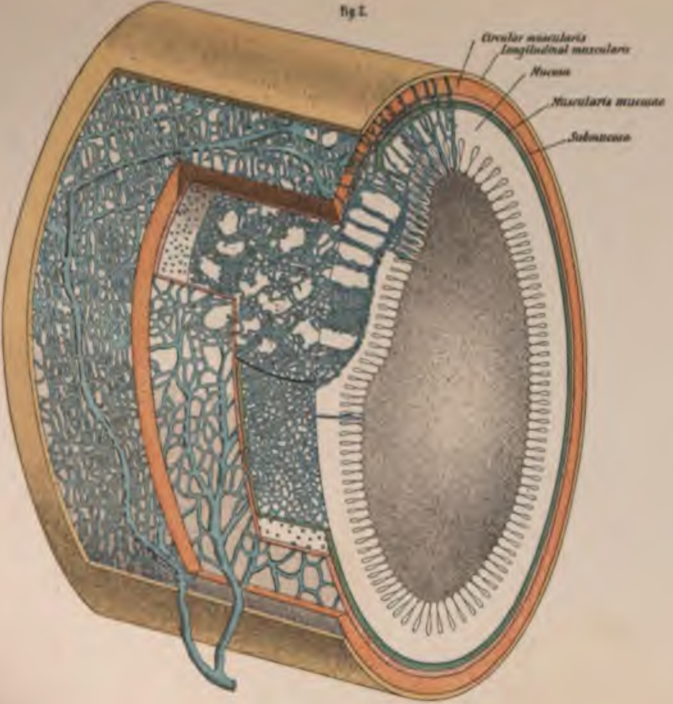
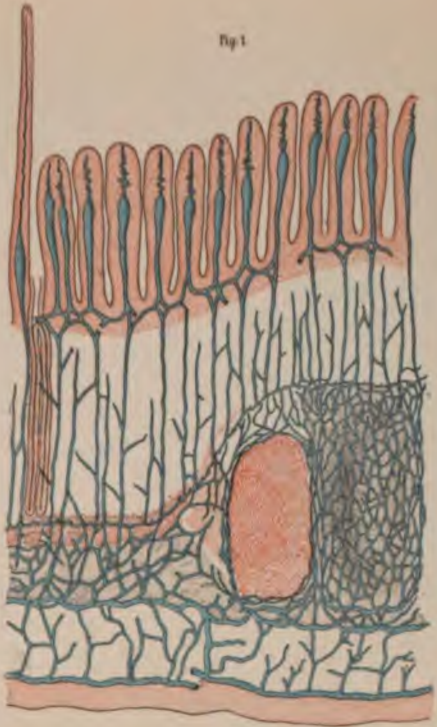


Fig. 1.



of the ileum; it varies very much in shape and size and also in its peritoneal investment, sometimes being simply covered or held down by a layer of peritoneum, and at other times having a distinct mesocecum, which admits of a considerable range of movement, sometimes even allowing it to be pulled over almost to the left iliac fossa. Usually the cecum is found in the right iliac fossa, but may be found higher up on the right side, even in contact with the under surface of the liver, or occasionally, according to the length of its mesenteric attachment, may hang down into the pelvic cavity.

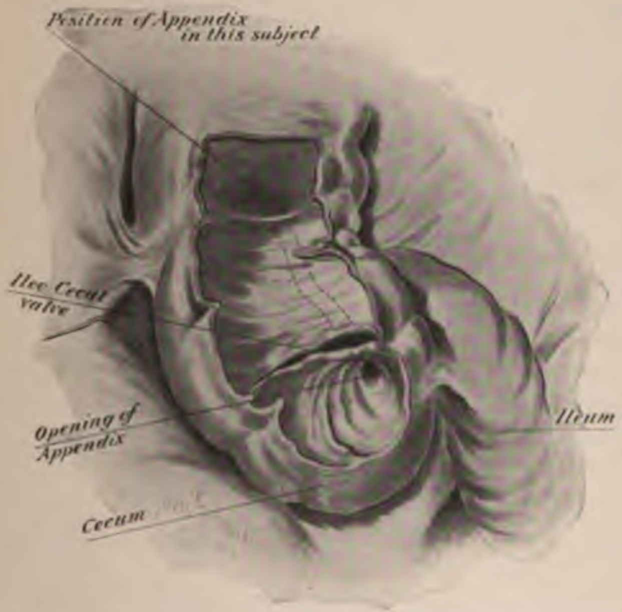


FIG. 3.—ILEOCECAL VALVE, OPENING OF APPENDIX.—(*From dissection in anatomical laboratory of the University of Maryland.*)

The Vermiform Appendix.

Attached to the lower end of the cecum is a tubular prolongation, the vermiform appendix, the walls of which are similar in structure to these intestines, its lumen being continuous with that of the cecum and colon. In length the appendix varies from two and one-half to six inches, though even longer ones than this have been observed. Its position varies in accordance with the position assumed and attachments formed by the cecum in the development and transposition of this portion of the intestinal canal in fetal life. Usually it is found in one of two positions—either turned upward or outward around the outer border of the cecum, or hanging down into the pelvis across the iliac vessels. Sometimes it may turn upward behind the ileum and along the inner side of the cecum, being in this case retroperitoneal. This position is shown well in the illustration (Fig. 3), which was drawn from a dissection.

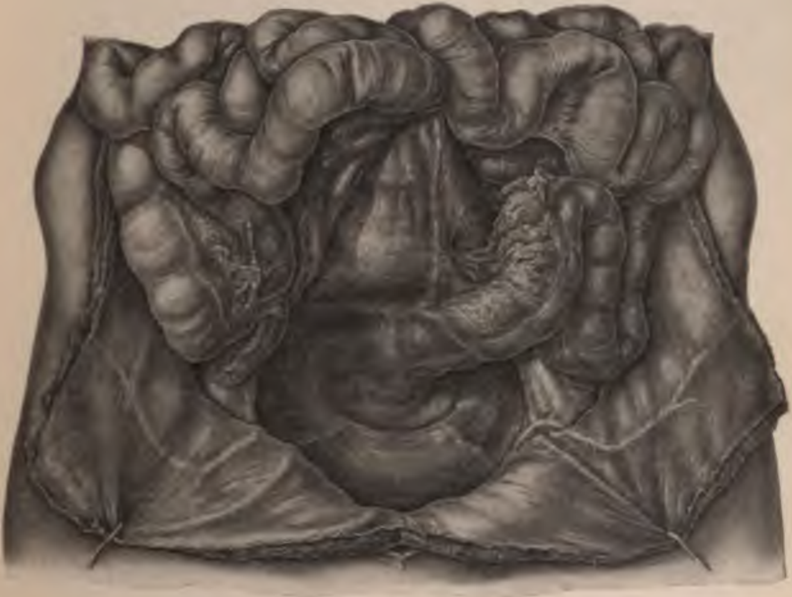
The peritoneal investment of the cecum varies from a complete investment to an almost complete absence, as in the case previously mentioned. Usually it has a complete investment of peritoneum with a more or less complete mesocecum.

The Ascending Colon.

The ascending colon passes upward from the cecum in the right lumbar region, in front of and in relation with the right kidney and descending portion of the duodenum, to the under surface of the right lobe of the liver. Here it makes a sharp bend to the left, —the hepatic flexure,—to become the transverse colon. The ascending colon varies very much in its peritoneal investment, being frequently covered only on its anterior surface, and we have all degrees of mesenteric attachment from this condition up to the formation of a complete mesocolon.

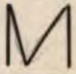
The Transverse Colon.

The transverse part of the colon usually crosses the umbilical region from right to left, slightly upward, and after it passes the front of the vertebral column, backward to the spleen, where it makes a sharp turn downward—the splenic flexure. The peritoneal investment of the transverse colon is complete, and is held in position by the transverse mesocolon. From the border of the transverse colon we have, hanging downward, a large fold of peritoneum,—the *great omentum*,—which may be spread out evenly over the front of the intestine, or rolled up along the transverse colon toward the splenic end.



PELVIC CAVITY OF MALE (SUPERIOR VIEW).

The cecum and appendix are plainly recognizable on the left; the sigmoid and rectum on the right. The relations of the male pelvic fossa are clearly represented. Close observance of the anatomical course of the sigmoid flexure makes the difficulty of its direct intubation and inspection intelligible.—(*From Deaver's "Surgical Anatomy."*)

Dislocations and Abnormalities of Position.—The transverse colon is subject to very many variations in position, and may cross the abdominal cavity at any point between the epigastric region and the pelvis—sometimes turning downward from the hepatic flexure, passing all the way to the pelvis, across the pelvis, and up on the left side to the splenic flexure, forming the so-called M-shaped colon; sometimes, as in a case examined by the writer quite recently, it passes from the hepatic flexure downward to the brim of the pelvis, from there upward and to the left, diagonally across the abdominal cavity to the splenic flexure, the first descending limb being very close to the ascending colon, thus:  Between the normal and these extreme conditions we have many varieties which are different in degree rather than different in kind. These cases are not always cases of acquired enteroptosis due to pathological conditions, but are congenital (see chapter on Enteroptosis and Coloptosis).

The Splenic Flexure.

The splenic flexure is slightly higher and more deeply seated than the hepatic. From the splenic flexure the descending colon passes down through the left lumbar region, lying in front of, or to the outer side of, the left kidney. It is more deeply seated than the ascending colon, and is subject to the same variations in its peritoneal investment.

It terminates in the sigmoid colon at about the crest of the ilium, where the sigmoid flexure begins.

The Sigmoid Flexure.

The sigmoid flexure is the lower portion of the colon, and its limits are usually given as from the crest of the ilium to the sacro-iliac articulation. In point of fact, the greater portion of the sigmoid flexure is normally to be found in the pelvis, the upper arm only of the S, so to speak, being found in the iliac fossa. The accompanying illustration, made from an original dissection after careful examination of many bodies with reference to the position of the sigmoid flexure, represents what is probably the normal arrangement. This projection of the sigmoid flexure into the pelvic cavity is to be remembered as a possible cause of obstruction of the rectum, especially when the sigmoid contains large quantities of feces, which would have a tendency to produce a distinct angulation at the point where the sigmoid and rectum join; or, if the sigmoid possesses an extra loop or twist, as emphasized by Curschmann (see chapter on Enteroptosis). The sigmoid flexure has a very distinct and large mesentery,—mesocecum, mesosigmoid,—which admits of a considerable range of movement of this part of the intestine.

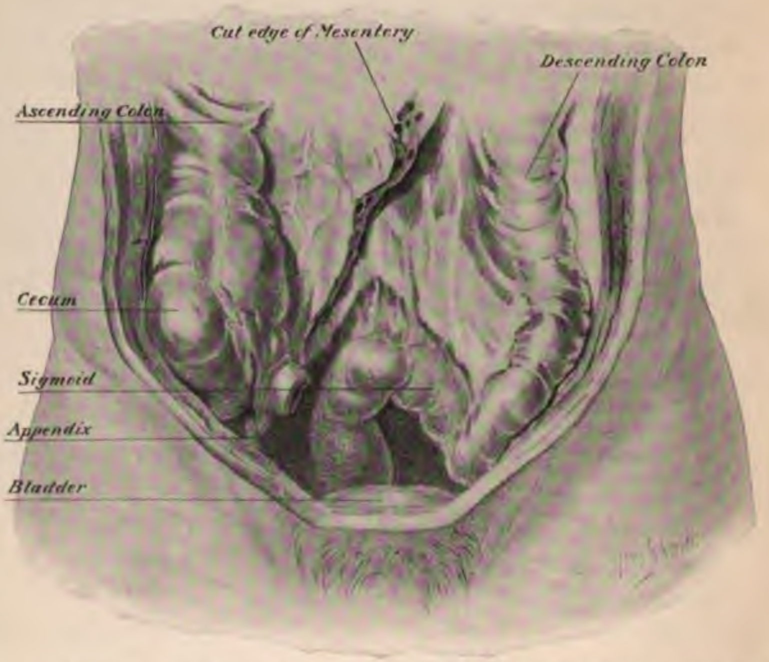


FIG. 4.—ASCENDING AND DESCENDING COLON, SIGMOID FLEXURE.—(*From dissection in anatomical laboratory of the University of Maryland.*)

Anatomical Aspect of Obstructions.

There are many conditions which interfere with the proper action of the intestines: for instance, changes in position, obstructions due to causes affecting the intestine independently of any extraneous circumstances—intrinsic, in other words; obstructions by bands and adhesions, and incarcerations in some of the natural openings of the abdominal cavity, due to either accidental or congenital conditions (see chapter on Intestinal Obstruction). Invagination is caused by irregular contraction of the circular muscular coat; this condition is said to be found often in people who die a sudden or violent death. The accompanying illustration was drawn from a specimen taken from the body of a negro man who was frozen to death during alcoholic stupor. In this case there were four invaginations, in every case the upper segment being pushed into the lower. The pathogenesis of invagination and its occurrence in cases of severe intestinal colic and spontaneous resolution are discussed in a separate chapter. When this condition occurs during life, the constriction of the intestine and the interference with the blood supply lead to rapid death of the constricted portion. Spontaneous recoveries have occurred, the apposing peritoneal surfaces becoming adherent and the constricted portion of the intestine sloughing off and passing into the canal; in other words, a spontaneous resection. Volvulus is a twisting of a coil of intestine upon itself in such a way as to cut off its blood supply. Its pathogenesis is given in a separate chapter.

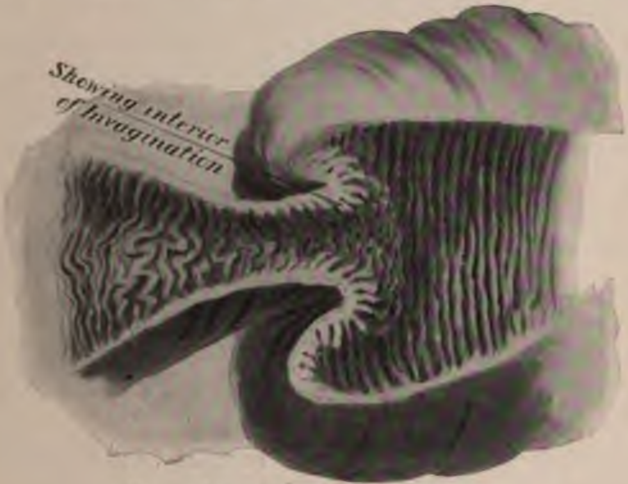
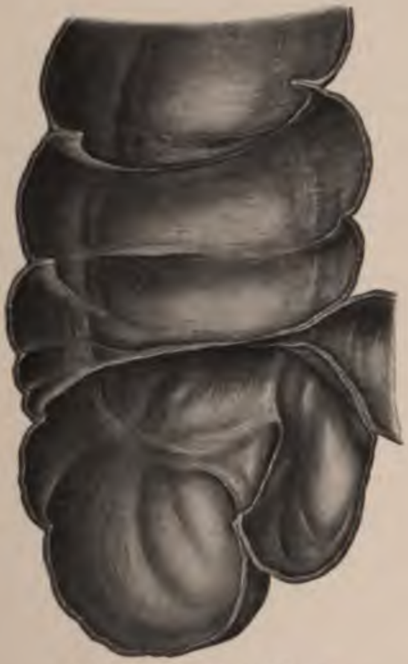


FIG. 5.—INTERIOR OF INVAGINATED ILEUM.

Obstructions Due to Bands or Adhesions.—Aside from the incarceration of the intestine in any one of the openings which exist normally in the abdominal walls, the intestine may be obstructed by adventitious bands or adhesions due to inflammatory processes. The great omentum may become adherent at some point,—occasionally the uterus,—and in this way form a band around which a coil of small intestine may become strangulated. There may be a slit in the great omentum through which a coil of small intestine may protrude and become strangulated, the margins of the opening acting as the constricting band. Perhaps the most prolific cause of interference with the permeability of the intestine is the persistence or failure to develop of some of the fetal remains—as, for instance, a condition which is frequently seen in negro children, where there is incomplete closure of the umbilicus, allowing of the protrusion of one or more coils of intestine. This condition varies in degree from the smallest opening of the umbilicus to a complete separation of the recti muscles. It is not necessary to go into all the various forms of hernia or protrusion of the intestine through some opening, natural or accidental, but we will mention the fact that a coil of intestine has been known to pass through the foramen of Winslow, forming a complete and fatal obstruction. Many cases of obstruction are due to persistence of the omphalomesenteric duct or the omphalomesenteric vessels. In order to understand the proper significance of these structures, we must recollect that in the weeks of fetal life there is a duct of communication between the vitelline sac and the intestinal tube, which duct should, under normal development, become completely obliterated, but which in some cases remains as a finger-like projection from the convex surface of the ileum at a greater or less distance from the cecum. This diverticulum or projection communicates with the intestine, and is lined with mucous membrane which is covered with villi. It may hang free in the abdominal cavity, or may be attached to the umbilicus. It is called Meckel's diverticulum. In length it varies from 2½ to 6 inches, and may be cylindrical in shape or expanded at its extremity into a cavity with fluid contents. Its distance from the cecum varies from 1 to 6 feet—usually about from 2½ to 3 feet. While Meckel's diverticulum may give no trouble, yet a considerable number of cases are on record in which it has acted as the constricting band and caused a fatal obstruction. In one case which occurred in the hospital of the University of Maryland a Meckel diverticulum nearly 6 inches in length was found attached to the umbilicus, and having its distal end



ILEOCECAL VALVE AND CECUM.
Colon is cut open, showing the edges of the valve and the sacculi of ascending colon and cecum.

expanded into a large bulbous sac, containing a considerable quantity of fluid. There were several coils of intestine twisted around the diverticulum, both intestine and diverticulum being strangulated.

The omphalomesenteric vessels sometimes remain as fibrous cords attached to the umbilicus or to some other point, and act as constricting bands.

The remains of the hypogastric artery may sometimes be a factor in the condition of obstruction or strangulation. Occasionally this artery, instead of lying close to the abdominal walls, runs upward to the umbilicus at some distance from the abdominal wall, having a distinct mesentery, which in this way forms a pouch on each side which might very well serve to guide the intestine to the inguinal or femoral ring; or, possibly, by stretching of its mesentery, form a pouch in which coils of intestines may become incarcerated.

Malpositions of the intestines are sometimes observed—as, for instance, in the case of complete transposition of the viscera, or due to some congenital defect in the development of the abdominal wall, as has already been mentioned: for instance, the failure to close at the umbilical point or median line of the belly, and occasionally the incomplete development of the diaphragm.*

There are no true diverticula from the large intestine. False diverticula are sometimes observed, which are usually dilations and extensions of one of the natural sacculations of the intestine (see chapter on Diverticula, etc.).

The large intestine is more prone to incomplete development than the small, not infrequently terminating in a blind sac. Allen † (*l. c.*) mentions a case, reported by Billard, in which the duodenum ended in a culdesac with fluid contents, and the small intestines beyond, barely admitted of the passage of a female catheter.

---

* Dr. S. P. Latane, while superintendent of the Lying-in Hospital of the University of Maryland, sent me for examination the body of an infant which had died apparently from apnea almost immediately after birth. The examination showed a congenital deficiency in the left leaflet of the diaphragm. The stomach and nearly all of the small intestines were in the left thoracic cavity, pushing the heart over to the right side, and compressing the lungs to such an extent as to cause death. For a full account and description of the omphalomesenteric remains the reader is referred to the admirable article of Fitz, printed in the "American Journal of Medical Sciences," July, 1884, in which are recorded more instances of variations in development and reduplication of portions of the intestine and the persistent remains of the omphalomesenteric structures.

† "Human Anatomy."

CHAPTER II.

PHYSIOLOGY OF THE INTESTINES.

The Chemical Processes Occurring in the Intestinal Canal.—The Action of the Succus Entericus, Pancreatic Juice, and Bile.

The proteids, fats, and carbohydrates which are propelled from the stomach into the duodenum are there digested by means of the pancreatic juice, the bile, and the secretion of the glands of Lieberkühn, generally called the "succus entericus." We have already given the gross outlines of the physiology of the intestinal secretion in the volume on "Diseases of the Stomach," and we will now limit ourselves to a more specific statement of proteolysis, amylolysis and adipolysis, and the action of the juices referred to, both considered singly as well as all together.

THE ALKALINE SUCCUS ENTERICUS.

This intestinal secretion is the product of the numerous tubular glands of Lieberkühn occurring in the small and the large intestines. The secretion of Lieberkühn's glands in the beginning portion of the duodenum begins considerably earlier than the entrance of the gastric chyme into this portion of the gut. Since the afflux of bile and pancreatic juice begins at once upon the ingestion of food into the stomach and as soon as these juices enter the duodenum, they stimulate the glands of Lieberkühn mechanically and chemically, and cause them to secrete.

Demant's observations on a patient with an artificial anus as a result of a herniotomy are of importance in throwing light upon the physiology of the secretion of Lieberkühn's glands. He found that the glands in the lower part of the ileum secreted very little or no juice when the stomach and upper part of the intestine were empty, but soon after meals this secretion invariably became considerably augmented. He was enabled to collect from twenty to thirty cubic centimeters of pure intestinal juice without touching the mucosa in any way; this juice was of alkaline reaction, and upon addition of acids a brisk effervescence resulted. In animals that were operated on according to the method of Thiry-Vella, it was found that Lieberkühn's glands secrete but very small quantities of juice when they are not stimulated, but that the secretion becomes more abundant by electrical, chemical, and mechanical stimulation (Quincke and Masloff).

It is not necessary for food to touch the intestinal mucosa directly in order to cause a secretion of this juice, but the glands of Lieberkühn in the lower portions of the ileum are reflexly set into operation by mechanical and chemical stimulation of the mucosa of the stomach and duodenum, so that the chyme already meets with an alkaline intestinal juice when it arrives in the lower portion of the intestines. This secretion reaches its highest point during the actual intestinal digestion, because the rapidly consecutive and energetic contractions of the musculature constantly bring new parts of the chyme in contact with the mucosa and the glands, thus causing frequent mechanical and chemical stimulations to these elements, and effecting a much stronger secretory activity.

The succus entericus is a pale-yellow, opalescent liquid of a strongly alkaline reaction, due to from 0.55 per cent. to 0.5 per cent. sodium carbonate, and effervescing strongly on the addition of acids. In addition to other organic substances it contains albumin and mucin. Later on we will give the evidence for assuming that the percentage of sodium carbonate in this juice and its consequent alkaline reaction varies with the acidity of the gastric juice. Concerning the origin, composition, and digestive action of the succus entericus we are confronted with contradictory statements. While most observers accept it as a characteristic secretion of the glands of Lieberkühn and claim to have found certain ferments in it, Hoppe-Seyler denies that conclusive proof of the origin of this juice from the glands of Lieberkühn has been furnished. The following ferments have been claimed to exist in the succus entericus:

1. According to Leube, Kölliker, Schiff, Thiry, and Masloff, a peptonizing ferment occurs in it which dissolves fibrins slowly and egg-albumen very slowly; it produces the solution of gelatin, and, according to Eichhorst, also prevents gelatin from solidifying.

2. According to Leube, Demant, and Grünert, a second unformed ferment is contained in it which transforms cane-sugar, lactose, and maltose into glucosids; it would thus apparently supplement the action of the pancreatic juice.

3. Finally, a diastatic ferment has been found in the intestinal juice, but, according to Quincke, Busch, Garland, Grünert, and Schiff, its diastatic action is much inferior to that of ptyalin or the diastatic ferment of the pancreatic juice. As the saliva and pancreatic juice have an energetic amylolytic action, and as the bile also contains a weak diastatic ferment, it is improbable that much diastatic work is left to be done by the succus entericus. Neither is it probable that the fibrin-digesting ferment of this juice ever has much work to do, because fibrin is very rapidly peptonized and absorbed in the stomach; nor can it be assumed that the sugar-inverting ferment of the succus entericus has an important function, because cane-sugar is effectively inverted by a ferment found in the gastric juice by Leube, and is also readily absorbed after inversion.

As the organism does not, as a rule, continue to produce such ferments for which it has no use, the doubt of some authors that these ferments are constant and unchanging constituents of this juice seems justified. The amylolytic action of ptyalin and the pancreatic juice ends in the formation of maltose and very little glucose, but the action of the inverting ferment of the succus entericus goes still further in its production of glucose; it might be added that, according to some authors,—Bourquelot, for instance,—inversion of maltose into dextrose is effected by intestinal bacteria.

Lieberkühn's glands of the large intestine do not form any diastatic ferment, and in this portion of the intestine such a ferment would find little work to do.

In stenosis and occlusions of the pancreatic and common gall-duct it is possible that a portion of the proteolytic and diastatic ferments which accumulate in the excretory bile- and pancreatic ducts may pass over into the blood, and thereafter be excreted in the succus entericus. In this way a partial adjustment of the loss of the normal secretions may be effected. The salts of iron, of lithium, and of the halogen elements and other substances, as well as the inorganic salts which are taken in the food by men and animals, are, under certain conditions, excreted again in the succus entericus after absorption. This has been demonstrated in patients suffering with intestinal fistulæ from whom the succus entericus could be collected. The most important function of the intestinal juice is carried on by virtue of the large percentage of sodium carbonate. It is due to this salt that the reaction of the intestinal mucosa is alkaline at an early stage in digestion. This reaction is maintained to the end of digestion. During the neutralization of the HCl, which is combined with albumin, by the sodium carbonate of the succus entericus and the pancreatic juice, considerable nascent CO_2 is liberated, which facilitates digestion mechanically by breaking up and forcing apart the solid portions of the chyme.

When the total acidity of the chyme increases, which is caused mainly by the gastric HCl and by the small amounts of organic acid formed during digestion,—lactic, acetic, and butyric acids,—the acids of the bile, and also the acids normally ingested with the food, then the secretion of the intestinal juice becomes more abundant. This is most likely due to a chemical stimulation of the glands of Lieberkühn (Thiry and Quincke). It is believed that the secretion of alkalis in the intestinal juice far exceeds in amount that excreted in the bile and the pancreatic juice. It is probable, also, that the alkaline reaction of the chyme in the large intestine is a result of the abundant excretion of intestinal juice; in the small intestine the chyme remains acid even after the addition of pancreatic juice and bile. During the second period of gastric digestion, when the gastric chyme contains large quantities of free HCl, it is assumed that the chyme in the duodenum is distinctly acid from this cause; but during the first stage of gastric digestion, when the HCl is combined with proteid, the acid reaction of the duodenum is due to the fatty acids liberated by splitting up the neutral fats by pancreatic juice and bile. These fatty acids are partially saponified by the alkalis of the pancreatic and intestinal juice, or are emulsified by these secretions and absorbed. Before the duodenum contains chyme, the reaction of its contents is the same as that of the mixed duodenal juice (pancreatic, intestinal juice, and bile). In forming a conception of the nature of digestion in the duodenum, it must be borne in mind that the gastric chyme is not expelled into this part of the bowel all at once, but gradually and in small portions, and that these meet more or less of the alkaline juices of the intestines already present; the conditions for neutralization of the HCl and organic acids are therefore favorable.

According to Bunge, the main work of the intestinal juice consists of emulsifying the fats and fatty acids which become liquid at the temperature of the body; this emulsification can take place only in an alkaline reaction. During a normal, active peristalsis these liquefiable fats and fatty acids of the chyme come into frequent and immediate contact with the alkaline and intestinal mucosa, so that gradually a considerable portion becomes emulsified by the alkaline succus entericus. This explains why the chyle vessels of the small intestine contain emulsified fat even at a time when the intestinal chyme is distinctly acid. It is necessary here to distinguish very carefully between the alkalinity of the mucosa and the alkalinity of the chyme. The mucosa may be distinctly alkaline and the chyme distinctly acid at the same time.

It is evident, also, that the saponification of the fatty acids, which are formed by the splitting-up of the neutral fats under the action of the pancreatic juice and bile, as well as the proteolysis and amylolysis, by means of the trypsin and amylopsin, is very much enhanced by frequent contact with the alkaline intestinal mucosa.

THE PANCREATIC JUICE.

Concerning the history of investigations on and chemical constitution of pancreatic juice, we refer to chapter XI, "Diseases of the Stomach," pages 57 to 60. The secretion of pancreatic juice, according to recent investigation, is a continuous one. After completion of duodenal digestion its formation does not cease, but the secretion accumulates in the excretory ducts of the pancreas. Mechanical and chemical irritation of the gastric and intestinal mucosa incites the pancreas into activity reflexly. It is also probable, for reasons stated elsewhere, that the alkalinity of the pancreatic juice increases with hyperacidity. This juice is a transparent, colorless, and odorless liquid, strongly alkaline, and so rich in albumin that it solidifies on boiling; it contains 0.43 to 0.46 per cent. of sodium carbonate, and effervesces on the addition of acids. It is not known how much of this secretion is formed in twenty-four hours; but, according to J. P. Pawlow (Wratsch, 1893, S. 1232, Russian), a dog secretes as much pancreatic juice in twenty-four hours as is necessary to neutralize his gastric juice. If the pancreatic juice is made to discharge externally by artificial fistulæ, the animal is poisoned by the hydrochloric acid of its own gastric juice. This juice, so far as we know at the present time, contains three, probably four, distinct, unorganized ferments: (1) A diastatic ferment known as amylopsin; (2) a proteolytic ferment to which Kühne gave the name of trypsin; (3) an adipolytic ferment known as steapsin. (4) According to W. D. Haliburton and T. J. Brodie ("Jour. Phys.," vol. xx, p. 106), pancreatic juice possesses a milk-precipitating substance, causing a granular precipitate in milk at from 35° to 45° C. There is no solidification, however, until the milk cools, when it sets to a coherent curd. On warming, the curd is broken up and the milk assumes its granular fluidity. The granular precipitate produced by pancreatic juice seems to be intermediate between casein and caseinogen. The ferments of the pancreas are not formed as such in the cells of the glands, but as proenzyme or zymogens, which, according to Heidenhain, are rapidly converted into the active ferments.

Amylopsin, the diastatic ferment, was first demonstrated in the pancreatic juice by Valentin in 1844. It converts raw and boiled starch, through the various stages of amylolysis, into maltose and glucose in a most energetic and rapid manner. According to von Voigt, it is capable of transforming glycogen into grape-sugar; and, according to Schmulewitsch, it is even capable of dissolving cellulose. The transformation of starch by amylopsin will occur in an alkaline, neutral, and acid reaction, and, according to Fleischer and Meltzer, it may even take place in the presence of small amounts of free HCl. Large quantities of organic acids—fatty, lactic, and acetic acids—do not appear to impede the activity of this diastatic ferment.

Trypsin.—The proteolytic ferment was first discovered by Claude Bernard in 1846. Trypsin acts best in a neutral or an alkaline reaction, but its action is not arrested by an acid reaction, and, as Fleischer and Meltzer have pointed out, not even by small quantities of free HCl. Organic acids, particularly fatty acids, when present in large quantities do not prevent tryptic digestion.

According to Kühne, the action of trypsin on albumin occurs in the following manner: The chemically very complex molecule of albumin contains two nitrogen-holding groups; one of these is transformed by trypsin into peptone directly. In the presence of alkalis this peptone combines with them, forming sodium and potassium peptone, for peptone can combine as well with alkalis as with the acids, and, according to Pall, can form numerous double salts—for instance, with iron, mercury, and potassium iodid. The second group of the albumin molecules undergoes decomposition and transformation, during which leucin, tyrosin, and asparaginic and other complex organic acids are formed. Glutin and gelatin lose their property of gelatinizing, and are converted largely into glutin and gelatin peptone, but other portions of these food elements may go through the same organic transformation as albumin.

Steapsin, the fat-splitting ferment, was first discovered by Claude Bernard, who asserted that the pancreatic juice contained an unorganized ferment which decomposed neutral fats into glycerin and fatty acids, and although he was opposed by Landweih and Duclaux, who held that this effect of the secretion was due to bacteria and not to a ferment, the latest and most reliable investigation of Boas, Fr. Müller, and Nencke confirmed the original experiments of Claude Bernard. This splitting-up of fat may occur in neutral, alkaline, and feebly acid media, and it is asserted by some that it may even take place in the presence of small amounts of free HCl. Organic acids in large quantities do not seem to impede this adipolysis. Not all the fat ingested is split up into fatty acids and glycerin. A portion is only emulsified by the intestinal juice. After the adipolysis a portion of the liberated fatty acids combines with the alkalis of the pancreatic juice, and most likely of the succus entericus, to form soaps. It is claimed that the emulsification of the fats which have not been saponified is very much aided by the presence of the products of saponification—viz., fatty acids and glycerin—and also by mucin. Adipolysis by steapsin appears to be a slow process. Berthelot found that fifteen grams of pancreatic secretion of the dog requires twenty-four hours to break down two decigrams of monobutyrin into butyric acid and glycerin. Blank allowed fifty grams of finely minced pancreas of the ox to work upon five grams of mutton fat for twenty-four hours; he found, in one experiment, that 20.7 per cent., and in another experiment 20.4 per cent., of the fat was split up.

Emulsification of Fat by the Pancreatic Juice.—Claude Bernard (1856) attributed to pancreatic juice the power of emulsifying the largest part of ingested neutral fats and fatty acids that are liquid at the body-temperature. Gad has shown that when oil contains free fatty acids it only requires contact with a 0.25 per cent. to 0.5 per cent. sodium carbonate solution to make a perfect milky emulsion. His experiments demonstrated that mechanical action was not necessary to make an emulsion. The 0.5 per cent. of sodium carbonate which is contained in pancreatic juice is not all available to aid in emulsification, because it is to a large extent neutralized by the free acid which descends into the duodenum from the stomach, by the acid albumin, and by the organic acids formed in this locality.

In my experiments, which were done with intubation of the duodenum after the stomach had been washed out with an alkaline solution after one of the double test-meals recommended by me, the contents of the duodenum were found acid. I drew the duodenal contents, in one case, exactly two hours and thirty minutes after the meal had been taken. In this case I knew from previous observation that the meal passed out of the stomach, leaving this organ empty after two hours. Sometimes no contents could be obtained from the duodenum. The average acidity found in this case in the duodenum, expressed in terms of acetic acid, was equal to 2.3 per 1000. The chyme drawn from the duodenum two hours and a half after the test-meal in a patient showing normal acidity of the gastric juice turned red Congo solution blue. It also gave the Boas resorcin test and the phloroglucin vanillin test. We will later return to the reaction of the chyme in the duodenum, but from the foregoing observations one would be disposed to assume that the alkali of the pancreatic juice is instrumental in effecting emulsification of fats only indirectly—viz., by neutralizing the acids of the gastric contents, and by producing a splitting-up of the other portion of the fats and formation of the soaps, which, in turn, as is well known, favor the emulsification of the remaining fat. J. P. Pawlow (" Die Arbeit der Verdauungsdrüsen," Wiesbaden, 1898) has given experimental evidence pointing to the fact that fat inhibits the secretion of HCl in the stomach and reduces the digestive power of the gastric secretion (*l. c.*, pp. 135 to 137). His work also goes to show that pure gastric juice, or a solution of HCl of the acidity of gastric juice, is a strong incentive to the secretion of the pancreatic gland (*l. c.*, p. 151). From this the following conclusions are justifiable: In cases of hyperacidity fats should not be prohibited, because they serve to reduce the intensity of the acid secretion of the stomach, and when the food consists only of carbohydrates and fats, a larger portion of the fats is capable of being emulsified by the pancreatic juice in the duodenum, because there is likely to be a smaller quantity of gastric juice secreted, with a less degree of acidity, which can be more easily neutralized by the alkalis of the pancreatic and intestinal juice. A portion of the sodium carbonate of the pancreatic juice is thus left over to aid in the emulsification of the fat. Pawlow discovered that a secretion of gastric juice was effected by simply showing food to his hungry animals; the mere passing of meat before the noses of dogs caused this secretion, which he termed the "*psychic gastric juice.*" Food that was swallowed by the dogs, no matter whether it was fat, carbohydrate, or proteid, generally produced this psychic gastric juice. Therefore in order to test whether the food was a stimulant to the secretion of the gastric glands it could not be fed to the animal, but had to be introduced into the stomach of gastrotomized dogs in such a manner so that the animal was unaware of it. In this way he discovered that proteids were incentives to gastric secretion and that fats and carbohydrates do not serve as stimulants for this purpose (*l. c.*, pp. 130 and 131). This confirms our view, which we have expressed on many different occasions, that the diet of hyperacidity should largely consist of fat and carbohydrates, and as the excess of HCl formed in this disease is very inimical to normal pancreatic digestion, the diet that we recommend in inhibiting the excessive formation of HCl will at the same time insure its own proper digestion in the duodenum. Practical and clinical experience teaches us that a diet of this sort is usually very well digested in hyperacidity.

THE BILE.

The human bile is a golden-yellow, sometimes an olive-brown, secretion; normal bile never has a green color. Its alkaline reaction is due to carbonates and phosphates. The secretion from the hepatic cells is a continuous one. Although the outflow into the duodenum ceases entirely after digestion has been completed in this part of the intestine, the secretion of bile, nevertheless, continues, and it is stored up in the intrahepatic bile-ducts and in the gall-bladder. In this way large quantities are ready, immediately after each meal, to meet the food as it enters the duodenum. The quantity secreted in twenty-four hours is said to vary from 400 to 1200 c.c.; the amount will vary according to the nature of the food, the amount of water introduced into or excreted from the body, the condition of innervation, of blood pressure, etc. Mosler states that abundant ingestion of water increases the quantity of bile by increasing its liquid consistency. A diet consisting of meat and very little fat, or exclusively of fat and carbohydrate, according to Fleischer reduces the amount of bile. The amount is furthermore diminished after loss of large quantities of water through the kidneys, skin, intestines, or stomach, after large hemorrhages with reduction of blood pressure, and during passive congestion in the portal circulation; in these cases the bile also becomes very much concentrated and more viscid, a condition which mechanically hinders its flow into the duodenum.

The following substances have been claimed to exert a stimulating influence on its secretion: the purgative neutral salts and mineral waters, the salts of the bile itself (Paschkis), olive oil (Rosenberg), certain alkalis, and, according to Schiff, colchicum, hyoscyamin, nicotin.

Constituents of the Bile.

The quantitative analyses of Hammarsten have given the following results:

Solid materials,	1.62	to	3.52
Water,	96.47	"	98.37
Mucin and coloring-matter,	0.27	"	0.91
Compounds of bile acids and alkalis,	0.26	"	1.82
Taurocholate,	0.052	"	0.203
Glycocholate,	0.204	"	1.61

Fatty acids,	0.024	to	0.136
Cholesterin,	0.048	"	0.16
Lecithin,	0.048	"	0.065
Fat,	0.061	"	0.095
Soluble salts,	0.676	"	0.887
Insoluble salts,	0.02	"	0.049.

In addition to the more important and characteristic constituents, the bile salts and bile pigment, cholesterin, mucin, and a diastatic ferment, the bile also contains small quantities of lecithin, soaps, fats, and inorganic salts.

Bile Salts.—These constitute 10 or 12 per cent. of the solids. They are mainly sodium salts of two distinct organic acids—namely, glycocholic and taurocholic acids. Hydrochloric and a number of organic acids liberate the bile acids from their combinations.

When the common gall-duct is obstructed, the bile and bile salts do not enter the blood directly, but through the lymph-vessels. Fleischl demonstrated in Ludwig's laboratory that the chyle contains bile acids when the common gall-duct is ligated, but when both the latter and the thoracic duct are ligated simultaneously, the blood remains entirely free of bile and bile constituents. These experiments, made upon animals, have been confirmed by pathological observations upon man. When the thoracic duct was compressed by neoplasms and the common bile-duct obstructed in the same individual, the characteristic symptoms of jaundice were absent. When the bile constituents enter the blood through the lymph-vessels, they exert a retarding influence on the rate of the heart-beat, the cholemia is severe, and symptoms of intoxication result. The bile salts are capable of producing symptoms of cholemia when they enter the blood ; they are then partly excreted in the urine, and another part returns to the liver in order to reenter the blood through the chyle. As long as the common gall-duct is permeable the bile acids are excreted only in the intestine, but from here they may in part enter the blood through mediation of the lymph-channels. In the urine the bile salts may be detected by Pettenkoffer's reaction, consisting of sulphuric acid and a 10 per cent. solution of cane-sugar, which is added to the urine and the mixture slightly heated, avoiding a higher temperature than 70° C. A purplish red color results. When bile salts are injected into the blood or under the skin, they are excreted with the bile through the liver.

The Bile Pigments.—Bilirubin and biliverdin are formed in the liver from hemoglobin. Virchow was the first to demonstrate that in old extravasations of blood the hematin becomes gradually reduced and is replaced by another pigment, which he designated hematogen. Jaffé, Salkowski, and Robin claimed that hematogen was identical with bilirubin, and Cordua, Quincke, and Langhans established the fact that when blood was injected under the skin into other tissues or into the peritoneal cavity of animals, bilirubin could be demonstrated in these localities. Naunyn and Minkowski contributed to the experimental evidence that bilirubin is formed in the liver from hemoglobin. On extirpating the liver from a goose, they subjected the animal, together with a normal goose, to the influence of arseniureted hydrogen. The urine of the operated bird half an hour later contained only hemoglobin, and that of the normal goose, biliverdin. The blood and the tissues of the goose without a liver were entirely free from bile constituents.

Cholesterin.—According to Naunyn, this substance is not formed by the hepatic cells, but from the epithelial cells of the bile passages. The bile contains from 1 to 2.5 per cent. of this substance. It is said to be increased by inflammation of the bile passages—cholangitis. It is insoluble in water, and kept in solution in the bile by virtue of the bile salts and soaps. The largest part of gall-stones is made up of cholesterin.

The Diastatic Ferment.—This is probably not a specific constituent of bile. Diastatic ferments occur in the urine and in the interstitial juices bathing the tissues, lymph, etc. The ferment of this kind which occurs in the urine and bile seems to be physiologically identical with amylopsin-zymogen of the pancreatic juice.

Concerning the function of the liver in rendering poisons that reach it through the blood harmless, acting as a barrier to intestinal autointoxication, etc., we refer to the chapters on Autointoxication and Intestinal and Hepatic Dystrypsia.

The Chemical Reaction of the Bile.

Bile by itself exerts little, if any, chemical effects of a digestive nature on food material. If we could study this excretion as we can study urine, it would probably be discovered that the chemical reaction of bile would vary as much as that of the urine.

Like urine, it might be found slightly alkaline, slightly acid, or amphoteric. The bile of oxen, dogs, and pigs, as well as human bile, reacts acid to phenolphthalein (A. Jolles, "Arch. f. d. ges. Physiol.," 1894, vol. LXVII, S. 1). Neumeister ("Lehrbuch d. physiol. Chem.," 2. Auflage, 1897, S. 195) is of the opinion that bile is alkaline, containing 0.2 per cent. sodium carbonate and 0.2 per cent. alkaline sodium phosphate. Jolles states that the bile of man and of the animals referred to is of a faintly acid reaction, not alkaline or neutral. Different specimens of bile from the same animal may show varying degrees of acidity. In titrating human bile with a decinormal solution of KOH, using phenolphthalein as an indicator, he found that one gram required on an average 2.36 milligrams to neutralize the acid salts. This subject has been recently investigated by Mr. E. W. Brown, and been reported in an article by R. H. Chittenden and Alice H. Albro ("Amer. Jour. of Physiol.," vol. 1, No. 3, p. 318). Mr. Brown found that pig's bile having an acidity equal to 0.5 milligrams NaOH had an alkalinity equal to 1.05 milligrams of decinormal HCl. The alkalinity, as indicated by lacmoid, was considerably greater than the acidity, indicated by phenolphthalein, in some cases; in other cases the reverse was found to be true. Rabbit's bile was found to present no measurable amount of acidity. Such alkaline reaction as bile yields with red litmus and lacmoid is ascribed to the presence of Na_2HPO_4 and NaH_2PO_4. These studies make it plain that the substances which give to bile a certain degree of alkalinity, to be detected by litmus and lacmoid, react acid to phenolphthalein at the same time. It is also emphasized that the so-called acidity and the so-called alkalinity of bile may vary in the same species under different conditions of diet.

Uses and Functions of Bile.

The subjoined is a synopsis of the uses and functions of bile:

1. Fats are brought into a fine, permanent emulsion by bile, just as by pancreatic juice.

2. Bile assists the fat-splitting effect of pancreatic juice (Nencki). Without bile, only 61 per cent. of tribenzoicin is decomposed by pancreatic juice; with bile, the total amount.

3. By its alkalinity it forms soaps.

4. It dissolves fats in minute quantities.

5. It dissolves the saponified alkaline bases which are insoluble in the juices of the intestines.

6. Animal membranes moistened with bile are more permeable to emulsified fats than membranes moistened with water (von Wistinghausen, Heidenhain); it therefore facilitates absorption.

7. Bile is a stimulant to the intestinal epithelial cells, and incites and maintains their proper function (Röhmann).

8. It is claimed that albuminous bodies and pepsin dissolved in the chyme are precipitated as a resinous, sticky deposit which adheres better to the duodenal wall and effects a better utilization of the albuminates thereby.

9. An inhibitory influence over putrefaction is ascribed to bile (Maly and Emmich).

10. An influence favoring an increase of the peristalsis of the intestine (Röhmann).

The emulsification of fats is favored by bile because it effects a splitting-up of the solid neutral fats and thereby forms soluble soap, which in turn advances the emulsifying process. As emulsification occurs only in an alkaline surrounding, it was thought by Fleischer that this process was favored by bile because it neutralized a part of the free HCl of the chyme. From what has been said concerning the variability of the chemical reaction of bile, it is evident that its power of neutralizing free acid is of questionable value. J. Munk has observed that a considerable amount of fat is decomposed in the intestines, even when the secretion of pancreatic juice into the duodenum has ceased. It has been assumed that the effect of bile in producing adipolysis is due to a fat-splitting ferment which has thus far not been discovered. The observation of Wistinghausen and Heidenhain, that fat passes through membranes moistened with bile without difficulty, whereas it passes through membranes moistened with water only under a high pressure, has been made use of to explain the manner in which bile favors the resorption of fats.

It is quite certain that fat is fixed and becomes adherent to membranes that are moistened with bile; this makes it plain why the emulsification of fats may occur in the duodenum when the reaction of chyme in the center of the mass may be acid. The bile, in fixing the fat to the surface of the villi, insures its admixture with the alkaline succus entericus; for although the center of the mass of chyme may be acid, the reaction at the duodenal wall will be alkaline; therefore emulsification can take place on the surface of the villi, and the fat, when once emulsified, can also readily be taken up by the epithelia. It is not known how much of the fats is absorbed in the form of fatty acids and soaps after adipolysis, or how much is absorbed in the form of minute fat globules after emulsification. I agree with Bunge in the opinion that the intestinal wall does not behave like a dead membrane during the digestive act; the mechanical behavior of a dead membrane in permitting the passage of fat has only a theoretical interest in explanation of fat resorption. From the experiments of Hoppe-Seyler and Heidenhain it is undeniable that the resorption of all the constituents of the chyme is a function of the protoplasm of the epithelia covering the villi and other intestinal surfaces. Thanhoffer claims to have observed vivid protoplasmic movements in intestinal epithelia moistened with bile, which he interprets as an evidence that the bile incites these cells to stronger activity, and thereby hastens the process of resorption. Voit, F. Müller, and Nasse have made very thorough investigations on the percentage of fats contained in the feces of human beings and animals; they found that it increased much more when the secretion of bile into the intestine was checked than after a cessation of the secretion of pancreatic juice. The influence of bile in increasing intestinal peristalsis is largely due to the thinning of the chyme caused by the abundant addition of this liquid secretion, thus preventing a concentration of the feces, which would retard the downward peristalsis. The mucin contained in the bile also favors the onward movement of the chyme.

The inhibitory influence of bile over putrefaction was supposed to be due to some physiological antiseptic in this secretion. Maly and Emmich claim to have discovered that the taurocholic acid in the bile is as effective an antiseptic as salicylic acid. By far the largest portion of the intestinal putrefaction occurs in the last portion of the ileum and in the colon; the investigations of Macfadyen, Nencki, and Sieber (*l. c.*) having conclusively shown that the fecal matter flowing from a fistula at the end of the ileum showed very little evidence of putrefaction. There has never been any serious controversy concerning the alkalinity in the contents of the colon, and as the taurocholic acid could not exist here as such, but would probably be rapidly converted into the taurocholate of soda, the position of Maly and Emmich is untenable. It is probable that the restriction of putrefaction which is ascribed to bile is due to an indirect action. Bile favors peristalsis and the rapid digestion and absorption of fats; this prevents the putrefaction under normal conditions. The absence of bile from the intestine, which occurs in obstructive jaundice, frequently produces sudden diarrheas in consequence of the excessive irritation of the intestinal mucosa by products of putrefaction. Undigested fat envelops the proteid portions of the chyme and prevents its proper digestion; therefore we may have albuminous decomposition in the absence of bile. Stadelmann observed that these attacks of diarrhea, as well as the intestinal putrefaction, will rapidly diminish when fat is excluded from the diet of icterus patients.

The Fate of the Bile Constituents.

The Bile Acids.—It is probable that the larger portion of these is absorbed as such from the jejunum and ileum. In dogs, in whom the bile flows outward and is lost entirely through gall-bladder fistulæ, the percentage of the bile salts in the secretion becomes less and less. On this ground it has been assumed that the bile acids which are absorbed are led back to the liver and there used again for the preparation of bile. The taurocholic acid which is not resorbed is decomposed into taurin and cholalic acid. The base is probably entirely absorbed by the intestinal mucosa, and used anew in the liver to form taurocholic acid with the resorbed cholalic acid, for this latter is not entirely eliminated with the feces: a portion of it returns into the blood. The glycocholic acid which has not been absorbed is in part eliminated with the feces; the other part is split up into glycocol and cholalic acid. Glycocol is absorbed, and cholalic acid is partially absorbed and partially eliminated in the feces. Mucin and cholesterin are eliminated as such in the feces.

Fate of the Bile Pigments.—A very small portion of these is resorbed and utilized again in the liver. The largest portion, it is believed, is decomposed by the putrefactive process into hydrobilirubin;

a portion of this is eliminated with the feces, and another portion with the urine. According to reliable investigators, the urobilin is identical with bilirubin. The normal color of the feces is usually attributed to the presence of hydrobilirubin, and the clay-colored stools of icterus patients are usually explained by the absence of this pigment. When the intestinal peristalsis is very much accelerated, a portion of the bile pigments is not converted into hydrobilirubin, and under these conditions bilirubin and biliverdin are found in the feces. It is probable that the bile pigments are converted into hydrobilirubin by the bacterial putrefaction. Zweifel has found that the meconium of the fetus contains no hydrobilirubin, but unchanged bilirubin and biliverdin. Rudolf Schorlemmer prefers the bichlorid of mercury test for bilirubin in the feces to any other ("Arch. f. Verdauungs-Krankheiten," Bd. VI, S. 283; see Harry Adler, "Examination of Feces").

According to a recent view of Bunge, the color of the stools in icterus is due to the presence of abundant undigested fat in the feces. He asserts that after extraction of the feces with ether the brown color of the normal stool will result. Bunge is of the opinion that the color of the stools is due to hematin and sulphid of iron, and not to hydrobilirubin. If this author's explanation concerning the color of icterus stools is correct, the color of the evacuations should become brown when fats are excluded from the diet.

THE RELATION OF GASTRIC TO DUODENAL SECRETION. PREPARATORY SECRETION IN THE DUODENUM.

The digestive and absorptive capacity of the stomach was formerly much overrated. In departing from our previous views we have recently come to another extreme opinion, which claims that the stomach is nothing more than a reservoir to receive the food and gradually transmit it to the intestine, and that the digestive and absorptive processes which occur in the stomach itself are so insignificant that they may be practically disregarded. The stomach in this sense serves the same object as the crop of birds.

Bunge has asserted that the chief office of HCl of the gastric juice is to prevent putrefaction and effect the destruction of large numbers of bacteria ingested with the food. Although this is partially correct and the HCl really serves to destroy the excess of bacteria and prevent gastric putrefaction and fermentation, the work of the HCl is not limited to this. There can be no doubt that important digestive functions are carried on by the gastric juice. In patients suffering from achylia gastrica on a nervous substratum, gastric fermentations are absent as long as the gastric contents are expelled into the intestine at the proper time. The motility of the stomach in these patients is usually good, which proves that the proper functioning of the musculature is not dependent on the presence of HCl. The chief object of the gastric juice is undoubtedly the digestion of albumin and proteid. This conclusion might be reached from the study of the gastric contents of patients suffering from hyperacidity. After a meal consisting of proteids, carbohydrates, and fats, when the contents are drawn in a few hours, one does not find any albumin or proteid food left undigested, but there will be numerous remnants of undigested carbohydrates. In achylia gastrica the reverse is the case; here the amylaceous foods are thoroughly digested by the ptyalin of the saliva, which has unrestricted power to act, because of the absence of the HCl.

The digestive and resorptive capacity of the intestine is more manifold and very much more extensive and prolonged than that of the stomach. Gastric digestion is limited to proteolysis and amylolysis. Practically there is no fat digested in the stomach, though, to be absolutely correct, it should be stated that from 1 to 2 per cent. of fat is split up into fatty acids and glycerin in the stomach, for Cash claimed to have discovered a ferment in the gastric secretion which is capable of splitting up that amount of fat. This may be due to bacteria, not to an unorganized ferment. In the intestine there is an extensive digestion of albumin and protein which has escaped peptonization in the stomach, effected mainly by the trypsin of the pancreatic juice. There is, furthermore, a splitting-up, emulsification, and saponification of large quantities of fat, and finally the amylopsin of the pancreatic juice, the diastatic ferment of the bile, and the ptyalin which has passed over into the duodenum from the stomach effect an energetic conversion of starch into sugar.

Preparatory Secretion of Mixed Intestinal Juices.—The nature and mechanism of the secretion of the mixed intestinal juice, consisting of bile and pancreatic and intestinal juice, differs considerably from that of the stomach. The gastric secretion starts, as a rule, after food has entered the stomach ; it is set up by the specific character, and the chemical, mechanical, and thermic stimulation of food, and ceases when the stomach becomes empty; but the secretion of the mixed intestinal juice begins at a time when no ingesta have as yet reached the duodenum. The accumulation of these juices begins simultaneously with the secretion of gastric juice (Claude Bernard, Rossbach, and Bernstein). The secretion of the liver is a continuous one, and more recently Heidenhain has demonstrated that the secretion of the pancreas is also continuous. The formation of the characteristic products of these organs continues during the period between the meals. The ready formed pancreatic juice and bile are stored up in the ducts of the pancreas and duct of Wirsung, and in the bile-ducts and gall-bladder respectively, so that these secretions are ready for action immediately at the beginning of duodenal digestion. As soon as solid or liquid food is taken into the stomach, the intestinal mucosa begins to produce large quantities of alkaline succus entericus, and an abundance of bile and pancreatic juice accumulates in the duodenum. During this time the duodenum is void of peristaltic movements, so that the discharge mixed intestinal juice is retained in this part of the intestine, which is facilitated by the characteristic horseshoe-shaped loop of this part of the gut (see illustration of duodenum, Fig. 6, p. 55), the end being brought to the same level as the beginning, being thus fitted to retain fluids poured into it from the liver, pancreas, and its own glands. When the first portions of the gastric chyme are expelled into the duodenum, this acid mass, by irritation of the sensory nerves of the duodenal mucosa, sets up vivid contractions of the musculature, which in turn effect an intimate admixture of the chyme with the mixed intestinal juice, and when this accomplished, an active downward peristalsis follows. The beginning of the intestine resumes its state of rest, and the afflux of mixed intestinal juice begins anew. The duodenum is thus capable of effecting a churning of its contents similarly to the stomach. It is assumed, on good grounds, that the secretion of the glands of Brunner and of Lieberkühn is augmented as a consequence of the irritation of the mucosa by the acid chyme. The more acid the reaction of the gastric chyme becomes during the progress of digestion, the more energetic will the resulting peristalsis be.

The anatomical structure of the wall of the duodenum and ileum is such that a narrow marginal zone of alkaline intestinal juice can exist close to the wall of the intestine, while an acid chyme may occupy the center, or lumen, of the bowel at the same time. If we, therefore, speak of any portion of the intestine containing acid chyme, it does not necessarily mean that the wall of the intestine may not be alkaline: this wall is continually pouring forth an alkaline succus entericus, which favors the splitting-up of fat droplets into fatty acids and glycerin. The so-called preparatory duodenal secretion ceases when the last portions of gastric chyme have been emptied into the duodenum and moved downward. The stomach is usually found empty in five or six hours, even after very large meals.

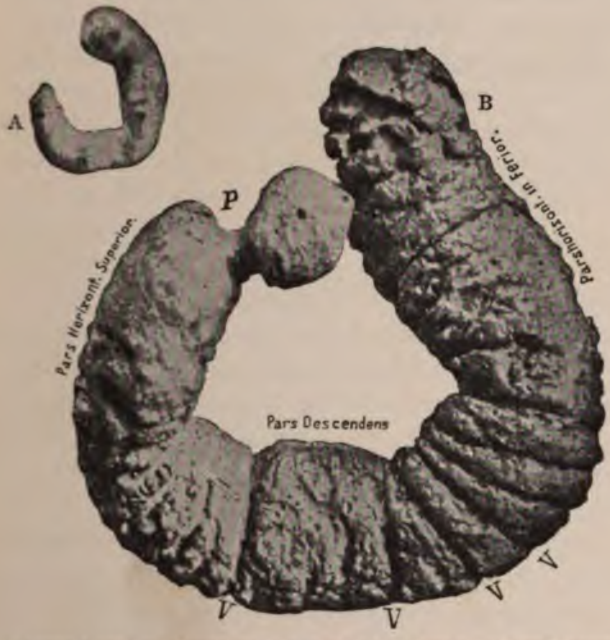


FIG. 6.—PLASTER CASTS OF DUODENUM OF INFANT AND ADULT.—(*From Museum of Harvard University.*)
A. Infant duodenum. B. Adult. V, V, V, V. Valvulæ conniventes. P. Pylorus.

In our observations with duodenal intubation (see Chap. XI) we found that the duodenum was generally empty in from two and a half to three hours after our test-meal, given on page 111 of "Diseases of the Stomach." It is not possible to say where the food mass may be at this time, but it is supposed to reach the ileocecal valve four hours after it has entered the intestine from the stomach. The chyme is retained for a much longer time in the large intestine (see chapter on Intestinal Peristalsis).

The preparatory secretion and accumulation of the mixed intestinal juice is of far-reaching importance for the normal digestion in the small intestine. When the gastric chyme brings sufficient HCl with it to make the duodenal contents acid from the presence of free HCl, digestion is not completely arrested: we simply have a continuation and extension of the gastric digestion to the duodenum in that case. Albumins are peptonized and starch is converted into maltose and dextrose by the diastatic ferment of the saliva if the amount of free HCl present is not more than 0.07 per cent. On the other hand, the digestion of fats, which can occur only in alkaline reaction, is inhibited in the center of the chyme, but not at the periphery, where the mucosa is alkaline.

The various intestinal ferments work most energetically in an alkaline reaction. Kühne found that the proteolytic activity of trypsin is destroyed by a prolonged exposure to gastric juice; he, therefore, concluded that a certain antagonism existed between these two enzymes; Boas, however, attributed the destruction of trypsin to the detrimental action of HCl, and Fleischer and Meltzer assert that trypsin is destroyed more rapidly by HCl and pepsin together than by HCl alone. If there were no preparatory secretion of alkaline mixed intestinal juice in the first part of the duodenum, a long time would elapse before the acid gastric chyme could be neutralized sufficiently to insure the digestion of various food-stuffs. In the second period of gastric digestion, when the percentage of free HCl in the gastric chyme is highest, the intestinal ferments would be destroyed if there were no accumulation of alkaline juice to neutralize and dilute the acid food mass coming down from the stomach. According to Boas, the ferments of the duodenal secretion are precipitated and lose their digestive power permanently when they are acted upon for a longer time by HCl. He also refutes the older conception that pepsin was precipitated by bile. The sudden entrance of considerable acid gastric chyme into the duodenum without being diluted and gradually neutralized would inevitably produce strong irritation of the intestinal mucosa, and energetic peristaltic movements which would propel the chyme downward so rapidly that normal digestion and resorption would suffer greatly. Thus the precipitation and destruction of the intestinal ferments, irritation of the mucosa, and excessive peristalsis are prevented by the preparatory secretion of mixed intestinal juice.

In view of these facts we may regard pyloric spasm occurring as a result of gastric hyperacidity not simply as a disease, but as a compensatory reflex mechanism, a preventive means retaining the excessively acid gastric contents within the stomach for a longer time than would otherwise be the case. In the mean while an abundance of mixed intestinal juices has accumulated in the duodenum, which serve to neutralize, at least partially, the acid gastric masses. According to Pawlow ("Die Arbeit der Verdauungsdrüsen"), the HCl of the gastric juice is a specific excitant to the secretion of the pancreas (*l. c.*, p. 152). In fact, he regards this as the most powerful excitant to the neurosecretory apparatus of the pancreatic gland (*l. c.*, p. 157). It has also been observed that the more energetically proteolysis is carried on in the stomach, the more prolific will be the secretion of bile and pancreatic juice into the duodenum. This latter is but a deduction from Pawlow's interesting studies, though the latter should be regarded rather in the light of an explanation, because the increased flow of bile and pancreatic juice with increased gastric proteolysis was known before Pawlow's publication. During normal gastric secretion the alkalinity of the blood from the acid salts of which the HCl is formed increases considerably; this must naturally be much more so in case of hyperacidity; consequently the percentage of alkalis in the intestinal juices will become larger with the increasing acidity of the gastric juice. In emphasizing that the preparatory secretion and accumulation of intestinal juice in the duodenum does not depend upon the presence of food, but begins as soon as food enters the stomach, we afford also a satisfactory explanation of the probability that the simultaneous secretion of acid gastric juice and alkaline intestinal juice prevents rapid and excessive variations in the alkalinity of the blood—such variations as might be dangerous to the organism and to the oxidization of the food materials in the blood, tissues, and cells.

This makes it also certain that the absolute loss or deficiency of gastric secretion may be a serious hindrance to metabolism, since this mutual chemical regulation which we have described between the stomach and duodenal secretions must necessarily be absent.

THE EFFECT OF GASTRIC ANACIDITY AND HYPERACIDITY UPON DUODENAL DIGESTION.

In the first period of gastric digestion the gastric chyme is expelled into the duodenum quite slowly and at long intervals ; in this period the gastric chyme does not contain any free HCl, but only acid salts. When the contents of the stomach reach the duodenum in this stage, they are naturally rapidly neutralized, and an alkaline reaction persists in the duodenum during this period. In the second period of gastric digestion the chyme contains free HCl, and the extrusions of food occur at much shorter intervals and in larger quantities. Therefore the duodenal chyme at this stage will be acid because the accumulated preparatory juice will have been partially consumed and will have a larger amount of acid to neutralize in order to reach that condition in which the intestinal ferments act most energetically. The greater the gastric acidity, the more time will elapse before the pancreatic and other intestinal ferments can become active.

The Effect of Anacidity.—In this condition very little or no proteids are peptonized in the stomach, but the ptyalin of the saliva remains very active during the entire gastric digestion. As HCl is a partial stimulant to the pancreatic and intestinal secretion and to the percentage of alkalis contained in them, the preparatory duodenal juice will be less energetic in its digestive powers, but the partial or complete loss of gastric proteolysis will yet be compensated by the tryptic digestion, for although the intestinal juice will be weaker in alkalis, it will have no acid gastric chyme to neutralize. It has been observed that intestinal putrefaction and fermentation increase with the reduction of HCl in the gastric contents.

The Effect of Hyperacidity.—When the gastric juice contains an excess of HCl, the duodenal preparatory secretion is not sufficient to neutralize it. The pancreatic and intestinal ferments work best in an alkaline medium, but they are also active in a neutral, or very faintly acid, medium. But when there is an excess of HCl pouring into the duodenum, the time in which the most favorable reaction is reached is considerably lengthened. A certain compensation is insured by the increased alkalinity of these juices when the gastric juice is highly acid. The loss or inhibition of tryptic digestion is partially counterbalanced by a more prolific conversion of proteids into acid peptones in the stomach; this proteolysis is still further increased in case the pylorus is closed off for a longer time from the duodenum by a pyloric spasm; but even if the duodenal secretions are insufficient to neutralize the gastric chyme, proteolysis does not cease, because it will continue as long as the chyme contains free HCl and pepsin. The digestion of fats and carbohydrates is very much delayed, however, particularly so as the conversion of starch into sugar can not occur in the stomach when the degree of free HCl has reached 0.12 per cent.

THE SPECIFIC IRRITABILITY OF THE VARIOUS PORTIONS OF THE GASTRO-INTESTINAL CANAL AND THE SPECIAL FOOD EXCITANTS TO THE DIGESTIVE SECRETIONS.

In almost all the modern text-books on digestive pathology the mistake has been committed to consider the mechanical stimulation of the food as the effective excitant of the digestive glands; this error is noticeable in some of the latest special works on the physiology and pathology of digestion. A most valuable contribution to the study of the work of the digestive glands has been published by Pawlow and his students ("Die Arbeit der Verdauungsdrüsen"). They gave the experimental evidence that the digestive canal is not universally irritable, and that it does not react without distinction to all mechanical, chemical, and thermic agents, but that the digestive glands react with distinction and regard for the peculiarity of each food, and that there is a certain specificity in the digestive secretions, which is controlled and determined by the amount and character of the work demanded. The first intimation that there may be specific excitants to various digestive secretions was given by Blondlot ("Traité analytique de la digestion").

The gastric glands, the pancreatic glands, the liver-cells, and the glands of Lieberkühn are controlled by a very delicate, sensitive, and complicated apparatus of innervation. Every new study of the digestive glands demonstrates a superb fineness of neuroglandular mechanism and a conformability to the object in view which are as beautiful as any of the recent studies in the structure and function of the cells of the cerebral cortex.

There are a number of excitants which are common to both the gastric and pancreatic cells; most important among these is the psychic moment expressed in appetite—the passionate desire for food, and, secondly, water. But both have their special excitants. The gastric glands are specifically and most powerfully stimulated by extractive substances contained in proteid bodies, meats, and the pancreatic cell is specifically and most powerfully stimulated by acids and fats. Alkalis inhibit the secretion of the pancreatic juice. The persistent addition of sodium carbonate and magnesia to the food in order to counteract the gastric hyperacidity reduces the secretory activity of the pancreas for a long time—the pancreatic juice becomes more and more inactive; this is the reason why the author has, in his volume on "Diseases of the Stomach," advocated the treatment of hyperacidity by local means (lavage with solutions of tannin, intragastric spraying with nitrate of silver), but, above all things, by diet. Fats diminish the secretion of the gastric juice, and thereby inhibit the digestion of proteids; for that reason the combination of fat and proteid food is generally indigestible to patients with deficient gastric secretion. The combination of a fat with a carbohydrate, such as we find in bread and butter, is not indigestible, because bread requires very little gastric juice and very little acid for its digestion, but the fat in the butter insures a prolific secretion of pancreatic juice for the digestion of the carbohydrate and proteid in the bread. Fat is indigestible only when combined with proteid in weak stomachs, because of the inevitable reduction in the amount of pepsin and HCl. Fat alone, without any other food, is not considered a difficult food to digest, because the inhibitory action on the gastric secretion can not then injure any other food, but rather favors the digestion and assimilation of the fat; therefore careful dieticians will exclude fat from the diet as much as possible whenever the gastric digestion is weak, but when the gastric digestion is characterized by an excess of H fats may actually serve as medicine, because they i secretion.

According to the investigation of G. Bruno (Inaugural Diss tation, St. Petersburg, 1898, Russian) on the secretion of bile i the digestive tract, it is certain that this secretion stands in the sa delicate and accurate relation to the various food substances, so as their stimulating power to the hepatic cell is concerned, as have observed with regard to gastric and pancreatic secretion.

So we may say that a diet [illegible]t contains only fats and carbo drates causes a secretion of the gastric juice deficient in free H and pepsin. Correspondingly, the alkalinity and quantity of t mixed intestinal juice becomes reduced. It appears that these t food substances, which are not altered by the secretion of gast juice, can not stimulate the gastric mucosa chemically. W mechanical stimulants only are applied to the gastric glands, th secrete a juice poorer in HCl and pepsin than when chemical stimulants act upon them, or as is the case when proteids reach the organ (Gmelin and Tiedemann).

CHAPTER III.

ABSORPTION FROM THE INTESTINES.

Tiedemann and Gmelin in 1820 compared the absorption process by the columnar cells of the intestine to the process of gland secretion. After the discovery of the laws of diffusion, attempts were made to apply them in explanation of the process of absorption. For a long time these physical views persisted, until it was shown by conclusive experiments that absorption, like other similar processes in the body, does not obey the laws of physical diffusion, but is selective in its character, and governed by the activity of the cells lining the intestines; thus our modern view is a recurrence to an older theory, with the exception, perhaps, that it is based upon broader experimental evidence. Absorption may, in fact, be compared to a kind of reverse secretion, for as the cells of a secreting gland take up materials from the surrounding lymph and from these elaborate specific products which are passed into the lumen of the gland as a secretion, similarly the columnar cells of the intestines take up certain products of digestion from the intestinal contents, by which they are bathed, and construct definite substances from these, which they pass into the lymph. The constituents of a secretion of a gland are specific and definite in their nature, and are most likely formed from definite constituents of the lymph taken up by the excreting cell to the exclusion of others. Similarly, certain materials only are taken up by the columnar cells of the intestine, and from these definite products are formed and passed into the lymph. In both cases the process is not a mechanical one,—not the passage of dissolved constituents through an inert membrane,—but it is distinctly a selective process.

The absorption of certain substances takes place in the stomach, but by far the greatest portion occurs in the intestines. Water is practically not at all absorbed from the stomach (Edkins and von Mering), but alcohol is readily absorbed. The absorption of certain substances in the stomach—for instance, sugar and carbohydrates—is favored by the presence of alcohol. The greater the concentration of substances, the more readily they are absorbed by the stomach, but the reverse holds for intestinal absorption—for instance, the rate of absorption of grape-sugar from the stomach increases up to a concentration of 20 per cent. (Brandl), but it is most rapidly absorbed from the intestine when its concentration lies at 0.5 per cent. As the concentration increases from this, the rate of absorption diminishes. The products of proteid digestion (Hoffmeister) and all forms of sugar are absorbed from the stomach to a greater or less extent.

Paths of Absorption.

There are two channels of absorption leading to the systemic blood current: one by the lacteals and the abdominal lymphatics to the thoracic duct, which empties directly to the subclavian vein; the other by the capillaries of the villi constituting the radicles of the portal system and eventually leading to the liver. These two channels have the following specific functions: the new substances formed by the action of the intestinal epithelial cells out of the absorbed products of digestion pass out of these cells into the lymphoid tissue of the villi underlying them. Thus the modified proteids and carbohydrates enter in solution into the lymph bathing the tissue of the villus, and in soluble form are absorbed from this lymph by the capillary vessels of the villi, thus passing directly into the portal circulation. But the fats leave the epithelial cells as fat globules, and are carried as such past the capillary network of the villus, to enter the lacteal situated in the axis of the latter.

The Absorption of Water.

According to von Mering and Edkins, water is practically unabsorbed from the stomach. Von Anrat and Tappeiner ligated the pylorus in dogs and found that considerable quantities of water were absorbed from the stomach, so that the results of the latter investigators and those of von Mering are contradictory. It is probable that von Mering's view is the more correct of the two, being based upon careful physiological experiments. It is probable, however, that the stomach of the healthy human being may absorb larger quantities of water when the blood is very much concentrated from any cause and great thirst exists. The absorption of water is probably most intense in the lower part of the ileum and throughout the entire length of the colon, causing the thin chyme of the upper part of the small intestine to become semi-solid, gradually assuming the consistency of feces. It has been shown by Heidenhain that the greater share of the water absorbed from the small intestine is taken up by the capillaries of the villi, and not by the lacteals, for when large quantities of dilute normal salt solution were injected into the small intestine, the rate of lymph flow in the thoracic duct was not markedly increased, unless the amount injected at one time forcibly distended the intestine. The absorption from the colon is also carried out by the capillary blood-vessels, so that practically all the water absorbed from the intestine is taken up by the blood circulation. The composition of the blood is little altered by the absorption of an excessive amount of water. The blood is not diluted to an extent corresponding to the amount absorbed; the absorbed water in such a case of excessive absorption passes first into the lymph which bathes the tissues, afterward to be eliminated gradually by the kidneys as the excess in the blood diminishes.

The Absorption of Salts and Soluble Constituents by the Columnar Cells Lining the Intestines.

The absorption of salts does not occur according to the laws of diffusion, but is a function of the protoplasm of the epithelium of the villi (Heidenhain). Thus it has been found that pure water is absorbed more rapidly than a ¼ per cent. solution of NaCl, and that a 2 per cent. to a 10 per cent. solution of NaCl is not absorbed at all, but causes the exudation of liquid from the intestinal wall; but during this exudation the percentage of salts in the liquid becomes less and less (Leubuscher). A 2 per cent. solution of potassium chlorid, which diffuses very rapidly through an animal membrane, is not absorbed from the intestine, but causes a transudation from the intestinal wall. Under normal conditions all substances which leave the epithelial cells in soluble condition are carried away from the lymph-spaces of the villus by the capillaries. Only those constituents pass into the capillaries which remain soluble after the action of the absorbing cells; fats do not belong in this class. When large quantities of sugar in concentrated solution are injected into the intestines, this is not the case. The work of absorption may become too great for the capillaries to perform in this case; part of it may then be absorbed by the lacteals.

The Absorption of Carbohydrates.

A large part of the carbohydrate food is absorbed from the intestines in the form of simple sugars, chiefly as levulose and dextrose, but it is probable that the absorbing cells are capable of taking up saccharoses and even colloidal carbohydrates, such as dextrin and starch, and converting these into simple sugars before turning them into the blood stream. The argument that dextrin is not directly assimilable because it is eliminated by the kidneys when it is injected in the circulation is not valid against its absorption as dextrin by the epithelial cell. The cell need not necessarily turn it over into the lymph in exactly the same form in which it received it. Sheridan Lea showed that the rapidity of diastatic action was much increased by dialysis, and the quantity of dextrin left unchanged in the dextrose largely diminished thereby; the logical deduction is that the more favorable the conditions are for the removal of digestive products, the more complete will be the conversion of dextrin into maltose. Musculus and Gruber came to the conclusion that the unchanged dextrin remaining after a prolonged digestion of starch with the diastatic ferment is not convertible into maltose by a fresh addition of the diastatic ferment after a complete removal of maltose produced by the first digestion; so that the failure of the ferment to convert the last part of dextrin into maltose can not be wholly due to the stoppage of its action by excessive maltose. After the transformation of starch into maltose has reached its final stage, there still remains a portion of achroodextrin unconverted into maltose; upon this remnant diastase has only a very slow action, but it is probable that although the digestive enzymes of the intestine are incapable of transforming all starch of the food into maltose and dextrose, a portion is absorbed as dextrin and changed by the absorbing cell into something else before it reached the blood stream. The percentage result when the reaction is completed, according to Sir William Roberts, gives in round numbers eighty parts maltose and twenty parts achroodextrin.

Röhmann has shown that not only sugar, but even starch, solution may disappear from a Thiry-Vella fistula with considerable rapidity, and as the succus entericus possesses only an exceedingly feeble diastatic action on starch, it seems that here the starch must be directly taken up by the columnar intestinal epithelium. According to Minkowski and Abelmann, 57 to 71 per cent. of starch is absorbed after complete extirpation of the pancreas. The brothers Cavazanni, under similar conditions, found an absorption of 47 per cent. With an intact pancreatic secretion it is not probable that any appreciable portion of the starch is absorbed as such. Ingestion of large quantities of solution of sugar leads to the appearance of sugar in the urine (Voit). It may also cause diarrhea; this is not the case when carbohydrate is introduced in the alimentary canal. The complete absorption and utilization of carbohydrates when taken in the form of starch are most probably due to the rate of assimilation and storage as glycogen in the liver, being able to keep up with the rate of absorption from the intestine.

An interesting experimental study of intestinal absorption has recently been published by E. Waymouth Reid ("Philosophical Transactions of the Royal Society of London," Series B, vol. CXCII, pp. 211–297).

He experimented especially with the absorption of serum, peptone, and glucose. In abstract his conclusions are as follows:

1. A physiological activity of the intestinal epithelium in the act of absorption is demonstrated by: (*a*) The absorption by an animal of its own serum, or even plasma, under conditions in which filtration into blood capillaries or lacteals, osmosis, and *absorption* are excluded. (*b*) By the cessation or diminution of the absorption of serum when the epithelium is removed, injured, or poisoned, in spite of the fact that removal, at any rate, must increase the facilities for osmosis and filtration.

2. The activity of the cells is characterized by a slower uptake of the organic solids of the serum than of the water, and a rather quicker uptake of the salts than of the water. The relation to one another of the absorption of these various constituents is variable in different regions of the intestinal canal (upper ileum, lower ileum, and colon).

3. No evidence can be obtained of specific absorptive fibers in the mesenteric nerves.

4. The state of nutrition of the cells is the main factor in their activity, and this is intimately associated with the blood supply.

5. In reduction of the rate of absorption without detachment of epi-

thelium the absorption of the various constituents of serum is reduced in the proportion in which they exist in the original fluid.

6. The activity of the cells may be raised by stimulation with weak alcohol without evidence of concomitant increase of blood supply.

7. The bile has not a stimulant action on the cells.

8. The cells exhibit an orientating action upon salts in solution (sodium chlorid especially). In a loop of gut with injured cells sodium chlorid enters the lumen from the blood at a time when it is being actively absorbed from a normal control loop in the same animal. This fact was first noted by O. Cohnheim.

9. The absorption of water from the gut is dependent upon two factors: (*a*) The physical relation of the osmotic pressure of the solution in the gut to the osmotic pressure of the blood plasma; (*b*) the physiological regulation of this difference by the orientating mechanism of the cells.

10. The chief factor in the absorption of peptone is an assimilation (or *absorption*) by the cells, while in the absorption of glucose, diffusion, variable by the permeability of the cells (and so probably related to their physiological condition), is the main factor.

11. By removal of the epithelium the normal ratio of peptone to glucose absorption is upset, and the value tends to approach that of diffusion of these substances through parchment paper into serum.

12. Absorption in the lower ileum is greater for the organic solids of serum, and less for peptone and glucose, than in the upper ileum. The relative absorption of water in the upper and lower ileum is variable.

13. The relative impermeability of the lower ileum to glucose disappears with removal of the epithelium.

14. Absorption in the colon is for all constituents of serum, and for peptone and glucose, far less per unit of measured surface than in the middle region of the ileum.

15. The normal relative excess of salt absorption from serum over water absorption, observed throughout the intestine, is most marked in the colon, and more marked in the lower than in the upper ileum.

16. Finally, it is suggested that the cell activity which causes serum to pass over to the blood is of the same nature as that involved in the orientating action of the cells upon salts in solution.

In a recent paper ("Zeitschr. f. Biol.," Bd. xx, 1899, S. 418) O. Cohnheim ascribes the transfer of fluids from the lumen of the exsected and surviving gut of the cat to oxygenated blood or normal saline solution bathing the outside of the viscus. In various ways it is shown that osmosis, filtration, and imbibition can be excluded without stopping the main effect, and, furthermore, that the presence of sodium fluorid causes a cessation of the process.

E. Waymouth Reid described a method in 1892 ("Brit. Med. Jour.," May 28, 1892) in which a "vital" transfer of normal saline solution to normal saline solution across exsected rabbit's gut at equality of hydrostatic pressure on the membrane was described.

The method had one advantage over that of O. Cohnheim, in that not only the absorption of fluid on the epithelial side, but the *output* on the serous side, was made evident.

Conditions of Absorption of Proteids.

The power possessed by the intestinal cells of absorbing various forms of proteid affords one of the best illustrations that this process is not one of mere physical diffusion. The products found toward the end of an artificial proteid digestion are distinguished from the proteids from which they originate by being slightly diffusible. To this fact great importance was at one time attributed, because it was thought that only proteids in a diffusible form were capable of absorption, and hence that peptonization was in all cases a necessary preliminary. It is now generally admitted that many forms of native proteid are capable of entering the epithelial cells without previous change by digestion or otherwise; and in those cases in which a proteid is incapable of direct absorption, a much less profound change than peptonization is sufficient to render it so—namely, conversion into acid or alkali albumin. Such an absorption of soluble proteid other than albuminose or peptone takes place not only in the small intestine, but in the large intestine and even in the rectum.

Voit and Bauer cleared a loop of small intestine of its contents as completely as possible by stroking it and separating it from the rest of the gut by double ligatures at each end. Various forms of proteid, in solutions of known amount and strength, were then injected into this loop; the intestine was replaced, and its contents were examined on killing the animal (cat or dog) some hours later. It was found that variable amounts of these proteids had disappeared; thus, in from one to four hours from 16 to 33 per cent. of white of egg had disappeared, and of syntonin from ox muscle, from 28 to 95 per cent. It might be supposed that the portion of proteid absorbed had been peptonized by traces of proteolytic enzyme which might be present in the intestine; but in the unabsorbed proteid remaining at the end of the experiment no albumose or peptone was found. Voit and Bauer also injected solutions of white of egg and sodium chlorid into the rectum of man and animals in a fasting condition, and found a marked increase (from six to eight grams in twenty-four hours) in the amount of nitrogen eliminated by the kidneys; in fact, an equilibrium of nitrogen metabolism may even be maintained in this way. Eichhorst, who confirmed these results, was of the opinion that no appreciable amount of peptonization takes place in the large intestine. Finally, the objection that the action is due to traces of enzymes has been disposed of by the observations of Czerny and Latschenberger, in a case of a fistula situated in the sigmoid flexure. The rectum was thoroughly washed out from the fistula, yet from 60 to 70 per cent. of the injected proteid disappeared in from twenty-three to twenty-nine hours.

This rectal digestion of injected proteids may, in the latter case, have been carried out by bacteria, for in the experiments of Czerny and Latschenberger the effect of bacteria is not eliminated. As a matter of fact, it is not possible to sterilize the colon or rectum of a living animal by any practicable means (Hemmeter, "Études expérimentales sur l'action digestive de l'intestines," "C. R. de XIII^e Congrès international de Médecine," Paris, August, 1900). My experiments lead me to differ from the opinion held by Eichhorst, Czerny, and Latschenberger,—viz., that no peptonization takes place in the colon,—for I have found proteolytic ferments present in sterilized fecal extract in considerable quantities (Hemmeter, "Ueber d. Verkommen von proteolytischen u. amylolytischen Fermenten im Inhalt des menschlichen Kolon," "Pflüger's Archiv f. d. ges. Physiol.," Bd. LXXXI, S. 152.

Assimilable and Nonassimilable Proteids.—Some forms of proteid, such as alkali albumin, prepared from the white of egg, and acid albumin, prepared from muscle, myosin, or fibrin, are directly assimilable—that is to say, when injected into the blood stream they are not excreted again by the kidneys; others, such as unchanged egg-albumen, caseinogen, and gluten, are, when injected, at once excreted in the urine. The latter forms must, therefore, under normal conditions, be changed during absorption before passing into the blood; but when excess of white of egg is present in the intestine, absorption oversteps the rate at which this change can take place, and a portion of the egg-albumen reaches the circulation unchanged. Under these circumstances this portion is promptly removed by the kidneys, and an alimentary albuminuria is the result, just as an excessive amount of sugar in the intestine produces alimentary glycosuria.

Relative Amounts of Proteids Absorbed in Different Forms.—It is evident, then, that absorption can take place in the form either of albuminose or peptone, of alkali or acid albumin, or even occasionally in that of native proteid; and the question arises, To what extent does absorption take place under natural conditions in each of these forms? Such a question is exceedingly difficult to answer by experiment. It is impossible to do so exactly by observation of the amount of each form of proteid present in the intestinal contents during proteid absorption, because the absorption is selective, and a substance present only in traces may be passing out of the intestine more rapidly (as it is continually formed) than another which is present in much larger quantity.

A rough estimate of the relative amounts of proteid absorbed as albuminose and peptone and that absorbed in other forms may be obtained from analyses of the intestinal contents during proteid digestion. Thus, Schmidt-Mülheim examined the contents of the stomach and intestine at varying periods during digestion of flesh in dogs; he found that the amount of proteid in solution, both in the stomach and in the intestine, was small at any given time, *but the amount present as albumose and peptone was always somewhat greater than that present in other forms.* When it is remembered that albumose and peptone are absorbed more rapidly than other proteids, this points to the greater part of the proteid being absorbed in these forms.

It is not known with certainty to what extent amido acids are formed from proteids in the natural course of intestinal digestion. The experimental evidence on the subject is somewhat conflicting, but the majority of the observers are of the opinion that but little proteid is absorbed as leucin and tyrosin, being nearly all absorbed as albumose or peptone, or even at a still earlier stage of proteolysis.

The only positive evidence as to the formation of leucin and tyrosin in natural digestion rests on the amounts found in the intestinal contents during proteid digestion. Such evidence can give a reliable estimate of the amount formed relatively to other proteid products only when the rate of absorption from the intestine of these various products is known.

Kölliker and Müller found only microscopic traces of leucin and tyrosin in the upper part of the small intestine of carnivorous animals, and none in the lower part. Kühne subjected fibrin to tryptic digestion in a tied-off loop of intestine into which the pancreatic duct entered, and subjected the residue to analysis after four hours. Leucin and tyrosin were found among the products, but the yield was small, and, moreover, the conditions in such an experiment are not quite comparable to those of natural digestion. Indeed, Kühne himself thinks it probable that the greater part of the peptone, being rapidly absorbed, escapes such a decomposition.

The relative amount of proteid decomposed in the intestine into products not ordinarily useful to the economy, such as leucin, tyrosin, and amido acids, and the amount absorbed and in various other useful forms, varies in wide limits with the condition of the nutrition of the animal and the amount of proteid ingested. Schmidt-Mülheim ("Arch. f. Anat. u. Physiol.," 1877, S. 549) states that in the proteid digestion of carnivora leucin and tyrosin are not formed at all or else in such small quantities as to cause no appreciable loss of nitrogen derived from the proteid food. But Sheridan Lea, on the other hand ("Jour. Phys.," London, 1890, vol. XI, p. 255), obtained a considerable amount of amido acids from the intestinal contents of a dog six hours after an abundant meal of lean flesh (one gram of pure leucin and three grams of tyrosin). This equals the total amount of these products obtainable from ten grams of dried proteids. It also indicates that a considerable percentage of proteid was being converted into amido acids and absorbed as such, because it may be assumed that leucin and tyrosin are absorbed as readily as the albumoses. Besides these substances, among other things the following chemical bodies are formed: butyric, valerianic, succinic, acetic, and lactic acids, ammonia, hydrogen sulphid, carbonic acid, hydrogen; furthermore, indol, skatol, kresol, and phenol. These substances are absorbed from the intestines also; they are found in the urine in combination with sulphuric acid, and are known as the *aromatic or conjugate sulphates*. The amount of these increases with the increase in the degree of decomposition. A part of these substances—it is true, some only in faint traces—is passed out with the feces. Foster, in his "Text-book of Physiology," fifth edition, part II, p. 476, states that such a decomposition of proteid (referring mainly to the formation of leucin, tyrosin, and amido acids) may serve as a safety-valve to the economy, diverting from the tissues an often unnecessarily large proteid metabolism. It is assumed that some of these products—leucin and tyrosin and their derivatives—are not perfectly useless, but, after their absorption, are of utility to the organism. Certainly the potential chemical energy of the proteid is lost to the economy, so far as the execution of certain physiological work is concerned. This has been advanced as an argument against the occurrence of this process to any marked extent, and if the production of these amido acids does occur, their usefulness is doubted. This subject is still under consideration, and can not be considered as definitely settled. The total amount of energy derived by an animal from its food is measured by the chemical form in which it enters and that in which it leaves the body, and a given portion of proteid, entering the body and then leaving it as urea carbon dioxid and water, will give up to the organism exactly the same store of energy, no matter what may be the intermediate steps by which it is reduced from one form to the other. Those that are of the opinion that amido acids and their derivatives are of utility to the organism assert that in the one case the energy is set free in the tissues, and in the other case the energy is set free in the intestines. In the second case the heat set free by chemical decomposition is communicated to the intestines and carried off by the blood to keep up the temperature of the body, thus sparing a certain amount of chemical energy which would otherwise have to be used for this purpose. It is my belief that such benefits as may be derived from communication of this heat are counterbalanced by the injurious influences to the general organism, which are exerted by the toxic products absorbed from the intestine. It is a common clinical experience that with the increased formation of phenol, kresol, indol, and skatol—the derivatives of tyrosin formed in putrefaction—there are evidences of deranged metabolism and nervous function, which again disappear when efforts are made to diminish intestinal putrefaction as far as possible.

Changes in Albumose and Peptone Occurring During Absorption.

When albumoses and peptones are injected directly into the blood, the organism reacts to them as to foreign bodies. They are speedily excreted by the kidneys. When injected in very large quantities, they are not excreted by the kidneys, because they produce such a fall in arterial blood pressure that the secretion of urine is arrested. Even in such a case the albumoses do not remain in the blood, but pass into the lymph (Shore, "Jour. Phys.," vol. IX, p. 549). Albumoses and peptone are thus proved to possess marked toxic properties; and, when injected in large doses, they may cause the death of the animal by a great fall in arterial pressure. They alter the nature of the blood so that it no longer coagulates, or does so very slowly, when drawn from the blood-vessels. They are not contained in the normal urine. Neumeister has found no traces of these bodies in the blood. These results prove that albumoses and peptones which are absorbed from the gastro-intestinal canal do not reach the general circulation as such, but are converted somewhere on their way into other substances which can harmlessly enter the circulation. It has been supposed that the seat of this modification during absorption was in the liver, but this has been disproved by Schmidt-Mülheim, Neumeister, and Shore. Ludwig and Salvioli isolated a loop of intestine in the dog and kept it alive by circulating warm defibrinated blood diluted with normal saline solution through it. They then injected one gram of albumose and peptone in a 10 per cent. solution, ligating the piece of intestine at both ends. After four hours the intestine contained about half a gram of coagulable proteid, and only traces of peptone, while the defibrinated blood used in circulating through the piece of intestine contained no albumose whatever; therefore the albumose must have disappeared in the intestinal wall. Neumeister states ("Lehrbuch d. physiol. Chem.," 1893, S. 251) that a solution of albumoses and peptones in defibrinated blood can be changed by mere contact with pieces of living intestine. The rapidity of the change is increased when a slow current of air is driven through the mixture, so that the pieces of intestine are brought in contact with the peptone and albumose. Contributions to this subject have been made also by Hoffmeister, Heidenhain, and Shore, the weight of the evidence going to prove that albumoses and peptones are modified during their passage through the epithelial cells of the intestine and stomach by action of the living cell protoplasm. It is not known what substances are formed from them, but it is generally accepted that the process is a conversion backward into coagulable proteid. A normal human being is capable of utilizing almost as *great a percentage* of the albuminous food eaten as a carnivorous animal; for beef it has been found that 2.5 per cent. of the nitrogen of the ingested meat is lost in man, and in the dog 2.2 per cent. According to Atwater, the same figures for fish-meat are 2 per cent. for man and 1.6 per cent. for the dog. A healthy human being can consume, digest, and absorb considerable quantities of albumin for a short time. J. Ranke was able to digest 420 gm. of albumin in 2000 gm. of meat; he utilized this completely, and excreted 86 gm. of urea during the experiment. The introduction of an excessive amount of proteid food makes great demands upon the digestive capacity of the stomach and intestines, so that the digestive power and energy of absorption of these organs very soon become exhausted. In addition to this, an early repugnance to excessive meat ingestion makes a continuance of this diet impossible. This is exemplified in the experiments of Ranke, who, on the third day, could utilize but 259 gm. of proteid, contained in 1280 gm. of meat.

The percentage of proteid food which is absorbed from the alimentary canal may be deducted from a comparison of the amount of nitrogen in the food with that of the urine and feces during the experimental period when such food is ingested. Such investigations have shown that vegetable proteid is absorbed much more imperfectly than that from animal sources. This is probably due to the envelop of indigestible cellulose which surrounds the vegetable proteid, and also to the fact that the nonnitrogenous part of the vegetable food, on account of its consistency and bulk, stimulates the intestines mechanically, thus causing increased peristalsis. As already stated, the percentage of nitrogen in meat and egg which appears unutilized in the feces of man amounts to 2.5 per cent., while that of milk varies from 6 to 12 per cent. The various vegetable proteids vary considerably in their absorbability; thus the proteids in the leguminous plants and cereals are absorbed as perfectly as those of animal origin.

The Digestion and Absorption of Fats.

Various theories have been propounded relating to the form in which fats leave the intestine. The only digestive secretion which contains an enzyme capable of exerting a chemical action on the neutral fats is the pancreatic juice. Its action consists in splitting the fats into fatty acids and glycerin. A certain amount of decomposition of fats into fatty acids has been claimed to occur in the stomach (Cash, "Arch. f. Anat. u. Physiol.," 1880, S. 323; also Marcet, "Proc. Roy. Soc. London," 1858, vol. IX, p. 306). The cause of this decomposition is probably to be sought in bacterial action during the first stage of gastric digestion.

The theories on fat absorption may be divided into two classes: (1) Those which hold that fats are absorbed in a particulate form, as emulsified fats or fatty acids, and (2) those which hold that fats are absorbed in solution, as fatty acids or soaps. We shall not go into consideration of the theories of the emulsification in the intestine, but simply consider the emulsion and solution theories of fat absorption separately.

According to their chemical constitution, fats are triglycerids—combinations of fatty acids with glycerin (a triatomic or triacid alcohol of the propyl series). They are nonnitrogenous food substances, containing only oxygen, hydrogen, and carbon. The main bulk of the fats introduced in the food is of animal origin; vegetable fats, like olive oil, are introduced only in small amounts. Fats consist of mixtures of olein, palmitin, and stearin, combinations of oleic, palmitic, and stearic acids with glycerin. The main mass of the fats is composed of fatty acids and glycerin in combination: nine-tenths of the former and one-tenth of the latter. The animal fats are almost entirely pure neutral fats, containing only very small quantities of free fatty acids—about 1 per cent.; it is said, however, that the liver fat and cod-liver oil contain about 10 per cent. of fatty acids. The splitting-up of fats may occur in a neutral alkaline and even in an acid reaction (Fleischer), and it is very much favored by the presence of bile. Von Nencki found that with the addition of bile to pancreatic juice two and one-half to three times as much fat is split up as without bile. One of the products of the splitting-up of fats,—the glycerin,—being very soluble and mixable with water, passes the columnar epithelium readily. It is possible that it is retained in the epithelium of the villi in order to be recombined with fatty acids to neutral fats. The free fatty acids are formed into fatty alkalis of soaps as far as the amount of alkali present in the mixed intestinal secretions permits of this process. The author is of the opinion that this amount of alkali which is capable of being turned into the intestine is limited, and upon experimental investigations he does not believe that all the fat that can be digested can be split up in this manner. We will return to a consideration of this question further on.

The sodium and potassium soaps are readily absorbed from the columnar epithelium, but the insoluble calcium soaps are probably passed out with the feces. After an abundant digestion of fat there are only traces of soaps in the chyle, and the percentage in the blood is not increased. It is assumed, for these reasons, that the fatty acids are converted back into neutral fats in the epithelial cells lining the intestines; according to some authorities, in the cells of the mesenteric lymph-glands.

The relation of the acids present in the duodenum to the process of adipolysis is not so thoroughly investigated as might be desirable. No doubt the free and combined HCl which enters the intestine from the stomach plays an important part in this process. The HCl which is not neutralized by the alkalis of the mixed intestinal secretion can be absorbed. It is not known whether such an absorption positively takes place, and if so, what effect it has on the soaps and fats absorbed into the epithelial cells. It is conceivable that HCl is also set free by the reconversion of peptone and acid albumin by the intestinal wall. Under the influence of this free HCl on the soaps, alkaline chlorids and fatty acids would be formed, and the fatty acids might possibly recombine with the free glycerin, spoken of before, to form neutral fats.

The fatty acids which are not bound to alkalis can be emulsified, according to Munk, under the same conditions as rancid fats. This author also holds that these fatty acids can be absorbed and take the place of neutral fats in the organism, being converted into neutral fats on their way to the intestine from the thoracic duct. According to J. Munk, 10 per cent. of neutral fats which are introduced into the intestine are absorbed in the form of free fatty acids in a state of emulsification. The objections that have been urged against the theory of Munk are the same as those urged against the theory of absorption of emulsified fats as such, which we shall consider presently.

As emulsification can take place only in an alkaline medium, it has been urged that this process could not take place extensively, because the chyme in the duodenum and upper part of the ileum reacts acid. We shall consider the reaction of the intestinal contents in a separate chapter. It is necessary to distinguish carefully here between the reaction of the intestinal contents and the reaction of the intestinal wall. The intestinal contents may be acid in the duodenum, while the wall is distinctly alkaline. Fat which comes in contact with the wall becomes emulsified and is absorbed, the wall being generally coated with bile and mixed alkaline intestinal secretion. The bile has a tendency to fix the fat and hold it to the surface of the intestinal wall, and, as is well known, membranes become more permeable to fat when they are saturated with bile. The rate of absorbability of fats varies with their melting-point: fats which are liquid at the temperature of the body are assimilated more rapidly and completely than fats which are not liquid at body-temperature. It is stated that Hippocrates knew of the indigestibility of foods containing stearin. He cautioned against the use of eel as a food because it contained too much of this substance. E. Voit discovered, by means of exact analysis of the feces, that stearin is the most indigestible of all fats, because it remains solid at the temperature of the body. He found that only 11.4 per cent. of this fat was digested, but of other fats, from 92 to 97 per cent. F. Müller and Munk found that the tallow-like fats from beef and mutton, which do not melt until they reach from 45° to 50° C., which is over the temperature of the body, are digested very well in the intestine (from 85 to 90 per cent.). Lard and butter are the most digestible of all ordinary animal fats.

The amount of fat that can be absorbed by healthy individuals varies considerably; it varies much more among diseased patients. In females the power of digesting and absorbing fats is considerably reduced during menstruation. The inability to digest fats which is found during icterus and also during the continued fevers persists for several weeks after convalescence. This should be considered in selecting the diet of such cases after convalescence. While a healthy adult may digest from 5 to 600 gm. of carbohydrates and about 420 gm. of proteids, the amount of fat that can be digested under the most favorable conditions is but 300 gm., according to

Rubner; the same is the case in dogs; any excess of fats introduced beyond this amount is excreted with the feces.

Emulsion Theories of Fat Absorption.—Direct observation of the amount of fats which leaves the intestine in the form of emulsified fats, compared with that which leaves it in form of soaps, glycerin, and emulsified fatty acids, is impossible in the present state of experimental physiology. Recent experiments tend to show that the statement of the absorption of emulsified fats is erroneous. In the author's opinion the objection to the emulsion theories of fat absorption are more apparent than real. Certainly the opposers of this theory—those who favor the conception of the solution theory of fat absorption—have not based their explanations entirely on facts proved by experiment. The objections are largely hypothetical; thus, in Schäfer's "Physiology," in which this subject is ably reviewed by B. Moore (vol. 1, p. 449), it is stated that the weak point in the emulsion theory of absorption always was to show how the fat globules got into the interior of the villi and made their way to the lacteal. Although the fat granules in the emulsion are of microscopic dimensions, they are still large compared with the dissolved molecules of serum or egg-albumen, which are unable to pass into or out of the intestine through the epithelial cells. Moore, therefore, asserts the necessity for a special kind of absorption by bulk by these cells and by process of even selected diffusion from solution; such an absorption by bulk is easily carried out by a cell of which the protoplasm is capable of free contraction, such as the ameba or leukocyte, but it is difficult to conceive how it can take place by a fixed cell, such as those which line the intestine. Right here we may urge against the argument of Moore that molecules of serum and egg-albumen can and do pass into the intestine through the epithelial cells in an unaltered condition (see the article quoted on pp. 436 and 437 of Schäfer's "Physiology," vol. 1). The author has gained the impression from experiments on the rectums of human beings and animals which were closed off from the colon by an inflated balloon (see "Digestion and Diet," by J. C. Hemmeter, "Maryland Med. Jour.," February 19, 1898, p. 328), that neutral fats can be absorbed from the rectum in the absence of digestive ferments and under conditions where the influence of bacteria can be reasonably excluded. Bacteria naturally produce decomposition in fats, and it is impossible to sterilize the rectum, even after shutting it off from the colon. But the unabsorbed fat remaining behind showed no traces of fatty acids or soap; this was suggestive that the fat had been absorbed as such. The experiments are not conclusive, but offered merely as a suggestion.

There is a distinct tendency in favor of the view that absorption of fats occurs almost exclusively only after saponification. The supporters of this theory would oppose the results of our experiments upon the rectum with the argument that the structure of the cells of the lining membrane of the rectum and of the intestine are unsuitable for the absorption of fats in the form of emulsion, and that fat granules have never been observed in the broad, striated border. The objection of J. Munk, that the amount of alkali necessary to split up 200 gm. of soap, which is the amount which can be absorbed in twenty-four hours by a dog weighing twenty-five kilos, would equal forty grams of sodium carbonate, which is three or four times the total quantity of alkali in the body ("Virchow's Archiv," 1884, Bd. xcv, S. 408), has been met by the assumption that alkali is again set free in the synthesis of fat from soap and glycerin within the cell in exactly an equal amount to that in which it was used up in the intestine, and that this alkali is returned again into the intestine to serve the same purpose over and act as a carrier to a fresh quantity of fatty acid in the form of soap into the cell. This explanation, of course, amounts to nothing more than meeting one hypothesis with another.

Solution Theory of Fat Absorption.—This theory is that neutral fats are split up by the action of steapsin into fatty acids and glycerin. The fatty acids unite, with a portion of the alkalis, with the mixed intestinal juice to form soluble soaps. The alkaline soaps and the glycerin are absorbed in solution by the columnar cells, and there synthesized back to neutral fats. The experimental evidence upon which this theory is based justifies the conclusion reached; at the same time it does not clearly disprove the possibility of the absorption of fat in form of emulsion. Heidenhain ("Arch. f. d. ges. Physiol.," 1888, Bd. XLIII, S. 513) still adheres to the emulsion theory of absorption, but does not explain the manner in which fat granules get into the epithelial cells. The bile, according to his statement, is indispensable in the process, favoring emulsification and thorough wetting and permeation of the membranes with fat emulsion. The fat globules pass on out of the columnar epithelial cells by means of protoplasmic contractions, according to this author.

The various theories as to the form in which fats enter the epithelial cells are summarized as follows:

Emulsion Theories.—(1) A *small* percentage of fat is split up into fatty acids and glycerin; the fatty acids unite with the alkaline bases of mixed secretions present in the intestine, and the rest of the fat is thereby converted into emulsion, which is absorbed by the columnar cells. (2) A *considerable* part of the fat is split up into fatty acids and glycerin and absorbed as emulsified fatty acids and glycerin, which are synthesized to neutral fats by the columnar cells.

Solution Theories.—1. *All* the fat is split up into fatty acids and glycerin; the fatty acids combine with the alkaline bases to form soluble soaps; these and the dissolved glycerin are absorbed in solution and synthesized to neutral fats in the columnar cells.

2. *All* the fats are split up into fatty acids and glycerin. The fatty acids are dissolved as such by the intestinal fluids (bile). These dissolved factions of the fat are absorbed by the columnar cells, and by these are synthesized again to neutral fats.

3. The processes under solution theories 1 and 2 probably neutrally replace each other to a variable extent in some animals, but in others absorption takes place entirely in the form of soaps.

LITERATURE ON THE ABSORPTION OF CARBOHYDRATES AND PROTEIDS.

1. Tiedemann and Gmelin, quoted by Heidenhain, "Arch. f. d. ges. Physiol.," Bonn, 1888, Supp. Heft, Bd. XLIII, S. 69.

2. Röhmann, "Arch. f. d. ges. Physiol.," Bonn, 1887, Bd. XLI, S. 411.

3. Busch, see "Virchow's Archiv," 1858, Bd. XIX, S. 171; Tappeiner, "Zeitschr. f. Biol.," München, 1880, Bd. XVI, S. 497; von Anrep, "Arch. f. Anat. u. Physiol.," Leipzig, 1881, S. 504; Meade-Smith, *ibid.*, Leipzig, 1884; von Mering, "Verhandl. d. Cong. f. innere Med.," Wiesbaden, 1893; "Centralbl. f. Physiol.," Leipzig u. Wien, 1893, Bd. VIII, S. 533.

4. Edkins, "Jour. Phys.," Cambridge and London, 1892, vol. XIII, p. 445; von Mering (*l. c.*); Gley and Rondeau, "Compt. rend. Soc. de biol ," Paris, 1893, p. 516.

5. Brandl, "Zeitschr. f. Biol.," München, 1892, Bd. XXIX, S. 277.

6. See F. Hofmeister, "Zeitschr. f. physiol. Chem.," Strassburg, 1882, Bd. VI,

S. 69; "Arch. f. exper. Path. u. Pharmakol.," Leipzig, 1855, Bd. XIX, S. 17; "Zeitschr. f. physiol. Chem.," Strassburg, 1881, Bd. V, S. 131.

7. Heidenhain, "Arch. f. d. ges. Physiol.," Bonn, 1888, Supp. Heft, Bd. XLIII, S. 53; 1894, Bd. LVI, S. 579.

8. Zawilsky, "Arb. a. d. physiol. Anst. zu Leipzig," 1876, Bd. XI, S. 161.

9. Schmidt-Mülheim, "Arch. f. Anat. u. Physiol.," Leipzig, 1877, S. 549; also 1879, S. 39; 1880, S. 33.

10. Von Mering, *ibid.*, 1877, S. 379.

11. Sheridan Lea, "Jour. Phys.," Cambridge and London, 1890, vol. XI, p. 226; see also pp. 255, 321, and 394.

12. Musculus and Gruber, "Zeitschr. f. physiol. Chem.," Strassburg, 1878, Bd. II, S. 177.

13. Brown and Heron, "Proc. Roy. Soc. London," 1880, p. 393.

14. Minkowski and Abelmann (Inaug. Diss., Dorpat, 1890; "Centralbl. f. Physiol.," Leipzig u. Wien, 1891, Bd. IV, S. 522) found, after complete extirpation of the pancreas, an absorption of from 57 to 71 per cent. of starch; the brothers Cavazzani ("Centralbl. f. Physiol.," Leipzig u. Wien, 1893, Bd. VII, S. 217), under like circumstances, an absorption of 47 per cent.

15. Otto, "Jahresb. ü. d. Fortschr. d. Thier-Chem.," Wiesbaden, 1888, Bd. XVII, S. 138; von Mering, "Arch. f. Anat. u. Physiol.," Leipzig, 1877, S. 413.

16. C. Voit, "Zeitschr. f. Biol.," München, 1891, Bd. XXVIII, S. 245; *ibid.*, Bd. XIX, S. 45; *ibid.*, Bd. V, S. 562.

17. Eichhorst, "Arch. f. d. ges. Physiol.," Bonn, 1871, Bd. IV, S. 570.

18. Latschenberger, "Virchow's Archiv," 1874, Bd. LIX, S. 174; see also Ewald, "Zeitschr. f. klin. Med.," Berlin, 1887, Bd. XII, S. 407; Huber, "Deutsch. Arch. f. klin. Med.," Leipzig, 1891, Bd. XLVII, S. 495.

19. Kölliker and Müller, "Verhandl. d. phys.-med. Gesellsch. zu Würzburg," 1856, Bd. VI, S. 499.

20. Kühne, "Virchow's Archiv," 1867, Bd. XXXIX, S. 130.

21. Foster, "Text-book of Physiology," 1889, fifth edition, part II, p. 476; part II, p. 513.

22. Neumeister ("Zeitschr. f. Biol.," München, 1888, Bd. XXIV, S. 277) caught the blood from the carotid in ammonium oxalate to prevent clotting; laked by shaking with ether; removed ether; saturated with ammonium sulphate; filtered; reduced filtrate by evaporating to a small bulk, filtered from time to time from drops of crystals; and tested in final filtrate for albumoses by the biuret test, with negative results. Control experiments showed that even a trace of albumose added to the blood intentionally could be easily identified. "Sitzungsb. d. phys.-med. Gesellsch. zu Würzburg," 1889, S. 65; "Zeitschr. f. Biol.," München, 1888, Bd. VI, S. 287.

23. Ploz and Gyergyai, "Arch. f. d. ges. Physiol.," Bonn, 1875, Bd. X, S. 536.

24. Fano, *ibid.*, 1881, S. 281.

25. Shore, "Jour. Phys.," Cambridge and London, 1890, vol. XI, pp. 528, 553, and 559.

26. Ludwig and Salvioli, "Arch. f. Anat. u. Physiol.," Leipzig, 1880, Supp. Band, S. 112.

27. Neumeister, "Lehrbuch d. physiol. Chem.," Jena, 1893, Th. I, S. 251; "Zeitschr. f. Biol.," München, 1890, Bd. XXVII, S. 324.

28. Pohl, *ibid.*, 1888, Bd. XXV, S. 31.

29. Czerny, "Beiträge z. operativen Chirurgie," Stuttgart, 1878, S. 141.

30. Ogata, "Arch. f. Anat. u. Physiol.," Leipzig, 1883, S. 89.

31. F. de Fillipi, "Deutsch. med. Wochenschr.," Leipzig, 1894, No. 40, S. 780.

32. Minkowski and Abelmann, "Jahresb. ü. d. Fortschr. d. Thier-Chem.," Wiesbaden, 1890, Bd. XX, S. 45.

33. Sandmeyer, "Zeitschr. f. Biol.," München, 1895, Bd. XXXI, S. 35 u. 40.

34. Bernard, "Compt. rend. Acad. d. sc.," Paris, tome XXVIII; "Arch. gén. de med.," Paris, 1849, series 4, tome XIX, p. 60; "Memoirs sur le pancreas," Paris, 1856.

35. Rachford, "Jour. Phys.," Cambridge and London, 1891, vol. XII, pp. 73 and 92.

36. Marcet, "Comp. rend. Soc. de biol.," Paris, 1857, p. 191; "Proc. Roy. Soc. London," 1858, vol. IX, p. 306; "Med. Times and Gaz.," London, 1858, new series, vol. XVII, p. 209. The extracts are taken from the last-quoted journal.

37. Kühne, "Physiol. Chem.," 1866, S. 129; 1868, S. 122.

38. Brücke, "Sitzungsb. d. k. Akad. d. Wissensch.," Wien, 1870, Bd. LXI, 2. Abth., S. 362.

39. J. Steiner, "Arch. f. Anat. u. Physiol.," Leipzig, 1874, S. 286.

40. J. Gad, *ibid.*, 1878, S. 181.

41. G. Quincke, "Arch. f. d. ges. Physiol.," Bonn, 1879, Bd. XIX, S. 129.

42. Von Frey, "Arch. f. Anat. u. Physiol.," Leipzig, 1881, S. 382.

43. Soxhlet, "Landwirthsch. Versuchsstat.," 1876, Bd. XIX.

44. Eberle, "Physiol. d. Verdauung," Würzburg, 1834.

45. Bunge, "Lehrbuch," 3. Aufl., S. 175.

46. Cash, "Arch. f. Anat. u. Physiol.," Leipzig, 1880, S. 323.

47. Altmann and Krehl, *ibid.*, 1889, Anat. Abth., Supp. Band, S. 86; 1890, Anat. Abth., S. 97; "Arch. f. Anat. u. Physiol.," Leipzig, 1890; Anat. Abth., S. 97.

48. Lebedeff, "Arch. f. Anat. u. Physiol.," Leipzig, 1883, S. 504.

49. Lewin, "Arch. f. d. ges. Physiol.," Bonn, 1896, Bd. LXIII.

50. Moore and Rockwood, "Jour. Phys.," Cambridge and London, 1897, vol. XXI, p. 74; also p. 58.

51. Von Thanhoffer, "Arch. f. d. ges. Physiol.," Bonn, 1874, Bd. VIII, S. 391.

52. Fortunatow, *ibid.*, 1877, Bd. XIV, S. 285.

53. Eberth, "Würzb. med. Zeitschr.," 1864, S. 23.

54. Arnstein, "Virchow's Archiv," 1867, Bd. XXXIX, S. 537.

55. Zawarykin, "Arch. f. d. Physiol.," Bonn, 1883, Bd. XXXI, S. 231.

56. Schäfer, "Internat. Monatsschr. f. Anat. u. Histol.," Leipzig, 1885, Bd. II, S. 6.

57. Munk, "Verhandl. d. Berl. med. Gesellsch.," March, 1879; "Arch. f. Anat. u. Physiol.," Leipzig, 1879, S. 371; "Virchow's Archiv," 1880, Bd. LXXX, S. 10; *ibid.*, 1884, Bd. XCV, S. 409; "Zeitschr. f. physiol. Chem.," Strassburg, 1855, Bd. IX, S. 568; "Arch. f. Anat. u. Physiol.," Leipzig, 1890, Supp. Band, S. 138.

58. Von Walther, *ibid.*, 1890, S. 329.

59. Radziejewski, "Virchow's Archiv," 1868, Bd. XLIII, S. 271; 1872, Bd. LVI, S. 211.

60. Perewoznikoff, "Centralbl. f. d. med. Wissensch.," Berlin, 1876, S. 851.

61. Will, "Arch. f. d. ges. Physiol.," Bonn, 1879, Bd. XX, S. 255.

62. Ewald, "Arch. f. Anat. u. Physiol.," Leipzig, 1883, Supp. Band; "Festschrift f. duBois-Reymond," S. 302; vorläufige Mittheilung.

63. Hoppe-Seyler, "Virchow's Archiv," 1863, Bd. XXVI, S. 534; Anmerkung.

64. Pettenkofer and Voit, "Zeitschr. f. Biol.," München, 1873, Bd. IX, S. 30.

65. Strecker, "Ann. d. Chem.," Leipzig, 1848, Bd. LXV, S. 29.

66. Latschinoff, "Ber. d. deutsch. chem. Gesellsch.," Berlin, 1880, S. 1911.

LITERATURE.

67. Quain's "Anatomy," eighth edition, 1876, vol. II, p. 363; "
1876, p. 194; "Internat. Monatsschr. f. Anat. u. Histol.," Leipzig,
"Arch. f. d. ges. Physiol.," Bonn, 1884, Bd. XXXIII, S. 513.

68. Wiemar, *ibid.*, Bd. XXXIII, S. 532.

69. Cohn, "Bull. Acad. de med.," Paris, 1856.

70. Schiff, "Jahresb. ü. d. Fortschr. d. Thier-Chem.," Wiesbaden, 1872, B
S. 222.

71. Von Mering and Minkowski, "Arch. f. exper. Path. u. Pharmakol.," L
1890, Bd. XXVI, S. 371.

72. Abelmann, Inaug. Diss., Dorpat, 1890.

73. Teichmann, "Mikroskop. Beiträge z. Lehre von der Fettresorption,"
Breslau, 1891.

74. Fr. Müller, "Zeitschr. f. klin. Med.," Berlin, 1887, Bd. XII, S. 45.

75. Bright, "Med.-Chir. Trans.," London, 1832.

76. Ziehl, "Deutsch. med. Wochenschr.," Leipzig, 1883, S. 538.

77. Le Nobel, "Deutsch. Arch. f. klin. Med.," Leipzig, 1888, Bd. XLIII, S.

78. Lewin, "Arch. f. d. ges. Physiol.," Bonn, 1896, Bd. LXIII, S. 171.

79. Noel Paton, "Rep. Lab. Royal College Phys. Edinburgh," 1891, vol. III, p

80. Wistinghausen, translation in "Arch. f. Anat. u. Physiol.," Leipzig, 1873,
137, by J. Steiner.

81. Gruper, "Arch. f. Anat. u. Physiol.," Leipzig, 1889, S. 505.

82. A. Bokay, "Zeitschr. f. physiol. Chem.," Strassburg, 1877, Bd. I, S. 157.

83. Hasenbroek, *ibid.*, 1888, Bd. XII, S. 145.

CHAPTER IV.

THE PERISTALSIS OF THE INTESTINES.

The intestines have three important functions, which, analogously to those of the stomach, are termed the motor, secretory, and absorptive functions.

A very intimate correlation exists between the sensory and motor functions in the intestines. Gad, Lüderitz, Nothnagel, and others incline to the view that the intestinal peristalsis is under constant and direct nervous influences, which are supposed to emanate from the plexuses of Auerbach and of Meissner. Engelmann, however, asserted that the origin of peristalsis is to be assigned to a process of excitation which extends from muscle-cell to muscle-cell directly, without the intervention of nervous influences.

A large number of capable physiologists and pathologists have contributed to our knowledge of this subject, but among the most important recent studies are those of Braam-Houkgeest and Nothnagel. The former first perfected a method by which the intestines of animals could be studied under warm normal salt solution, and to Nothnagel we are indebted for important researches concerning the differences in action of potassium and sodium salts when applied directly to the intestines, also concerning the action of certain narcotics on the peristalsis and on the processes and complications which develop in connection with acute occlusion of the bowel.

Under normal conditions, when the stomach and bowels are empty, during sleep, and in the fetus during the entire intra-uterine life, the bowels are in a state of perfect rest. This is sometimes spoken of as "aperistalsis." This condition of rest in the intestines persists even after the ingestion of food into the stomach. Even during the first stages of gastric digestion there is no intestinal peristalsis. During this period there is a copious secretion of alkaline bile, pancreatic juice, and succus entericus into the duodenum. It is well known that these secretions stimulate the wall of the duodenum mechanically and chemically; nevertheless, there are no peristaltic movements in the duodenum until the first portions of the gastric contents enter it. The explanation of this duodenal aperistalsis during gastric digestion has been explained by assuming that the movements are held in check by an inhibitory influence, emanating from the central nervous system and transmitted through the splanchnic nerves. Not until the first portions of the gastric contents have entered the duodenum do we observe active and rapidly consecutive alterations in form and position in the small intestine—the large intestine still continues in rest at this time.

The objects of intestinal peristalsis are the following: (1) The production of an intimate mixture of the chyme with the intestinal juices; (2) considerable neutralization of the acid gastric chyme by this admixture and by bringing it into contact with new surfaces continuously secreting the alkaline succus entericus; (3) to aid in the absorption of the soluble constituents of the chyme; (4) lastly, and most important, the propelling of the chyme downward, thereby creating room for new chyme coming out of the stomach, and forcing that already received over new areas of intestine constantly secreting their characteristic alkaline products.

Intestinal peristaltic movements may be classified as (1) physiological and (2) pathological. Braam-Houkgeest and Nothnagel distinguished three types of the physiological or normal movements: (*a*) The normal peristaltic movement; (*b*) pendulum movements; (*c*) rolling movements. All these movements owe their origin to alternating degrees and types of contraction of the longitudinal and circular muscular fibers.

The *peristaltic movement proper* consists of single expansions, caused by contraction of the longitudinal muscular fibers, immediately succeeded by a single constriction, effected by contraction of the circular muscular fibers. This produces a wavy peristalsis, which progresses toward the anus with moderate velocity. One such peristaltic wave extends only for a certain distance—never for the entire length of the small intestine. The contents of the bowel are propelled toward the rectum by this undulatory movement. S. Exner also observed that the haustra, or sacculi, of the colon are periodically drawn in and out in a regular manner during this peristalsis.

In the *pendulum movements*, one and the same piece of the intestine is moved to and fro without any demonstrable change of its lumen. These movements are restricted to very small portions of the intestine—generally not more than eight or ten centimeters participate in them. Nothnagel states that they are principally observed in the middle and lower portions of the small intestine when they are moderately distended with semi-liquid contents. There is no downward progress of the contents during the pendulous movements, except when they are combined simultaneously with peristaltic movements. The pendulous movements effect an intimate admixture of the individual constituents of the chyme among themselves and with the digestive secretions. They also favor repeated contact of continually renewed portions of the chyme with the intestinal mucosa, so that the resorption of food substances is considerably advanced by this movement. The duration of the pendulous movements may be fifteen minutes, after which they cease as suddenly as they began, and a prolonged period of rest ensues. The period of rest may extend from one to one and one-half hours, but in those portions of the intestine adjoining the cecum the periods of rest are much shorter than in the upper portions.

The *rolling movements* occur principally when a section of the intestine is extensively dilated by accumulations of gas and simultaneously contain very liquid chyme (Nothnagel). They lead to extensive changes of position of the intestine, and are, as a rule, accompanied by the peristaltic movements proper. The rolling movements propel the contents over a distance of from ten to twenty centimeters in a very active and energetic manner. The description of Nothnagel is particularly graphic: "Gas and liquid contents are propelled toward the ileocecal valve in lively haste, so that the fully distended intestinal portion seems to roll along like a rapidly revolving wheel, the effect being brought about by the circumstance that the circular constriction always runs closely behind the progressing and expanded loop. This peristalsis may be suddenly arrested at any portion of the intestine, as if by a jerk, and that too without any demonstrable external cause" (Nothnagel attributes the arrest to sudden nervous inhibition). After a varying pause the stormy rolling movements may again proceed onward. When one observes the normal intestinal movements under normal physiological salt solution kept at body-temperature during the period of intestinal digestion, manifold and alternating peristaltic movements are evident. While some portions of the intestine persist in absolute rest, others are in active movement. Two portions of the bowel distant from each other may be going through lively peristaltic movements, and the portion between these may be perfectly quiet. Then, again, this hitherto quiet portion may suddenly assume undulatory peristalsis while the two portions that were just observed in movement have come to rest.

Nothnagel considers the rolling movements the transition form to the pathological intestinal peristalsis. The peristaltic storms in the human intestine, which are accompanied by loud cooing and gurgling noises, but are painless (tormina intestinorum, Kussmaul), are attributed to this phenomenon. The peristalsis of the small intestine is very active and lively; that of the large intestine, comparatively slow and indolent. Accordingly, the chyme, after its entrance into the duodenum, traverses the small intestine rapidly, so that the entire contents after a large meal reach the ileocecal valve in from two and one-half to five hours. The much shorter distance from the cecum to the rectum is traversed in from twelve to twenty-four hours. It should be emphasized that one and the same peristaltic wave never traverses the entire length of the small intestine from the duodenum to the ileocecal valve under physiological conditions. Even the most stormy rolling peristalsis is arrested completely after it has traversed a limited distance.

The increased normal rate of peristaltic progress in the small intestine, in contradistinction to the slow and retarded movements of the colon, has been attributed to the greater chemical and physical irritation to which the small intestine is subject, for the chyme which enters the duodenum contains not only free HCl, but frequently hard and rough food particles. The HCl is gradually neutralized by the mixed alkaline intestinal secretions, so that the chyme in the lower portion of the ileum presents a neutral or even faintly alkaline reaction, while the larger food particles have become softened and disintegrated by the time the chyme reaches this portion of the intestine.

Experiments on the rate of the peristalsis of the small intestine and the colon, after stimulation of equal intensity, have, however, shown that the relations of the rate remain the same, that of the small intestine being active and that of the colon being slow. The nature of these rates of contraction appears, therefore, to be an inherent quality, characteristic of the neuromuscular apparatus of these two portions of the intestines. The slow and impeded peristalsis of the colon is of fundamental compensatory importance when, for some reason, the digestive capacity of the small intestine is injured; normally only 14.25 per cent., or, in other terms, only one-seventh of the total amount, of proteid ingested is left for digestion in the colon. This means that 85.75 per cent. is digested and absorbed from the stomach and small intestine. Under pathological conditions where intestinal digestion is much reduced, this relation may be exactly reversed, so that nearly six-sevenths of proteid digestion may fall to the colon. It has been observed under such conditions that food substances are almost completely absorbed, and the composition of the feces does not differ from the normal, provided the colon is in a healthy state. This extraordinary demand for work finally exerts a deleterious effect upon the colon. When increased irritation and irritability of the sensory and motor nerves of the colon occur simultaneously, the inhibitory provision, as shown in the slowing of its peristalsis, gives out, and sudden diarrhea may eventually supervene. Even the normal colon is capable of executing very lively peristaltic movements. This may be observed in the sudden diarrhea occurring in some healthy people after intense mental emotions, or also in those diarrheas associated with the various inflammations affecting the small intestine, and accompanied by increased motility of the colon. The slow peristalsis of the colon is usually not attributed to any anatomical peculiarity of the structure, but to a nervous inhibition, probably emanating from the nervous central organs. The rate of circulation through the intestinal walls is much accelerated by frequent and energetic contractions of the musculature. Accordingly, the circulation in the walls of the colon must be proportionately slower than in the walls of the small intestine. This explains the different pathological results observed in the large and small intestine after diseases of these organs, or of other organs which cause a passive congestion in the gastric and intestinal blood-vessels.

Under physiological conditions, all intestinal movements are caused by irritation of the contents, chyme, feces, or gas. The very intense rolling movements are attributed to distention of individual loops by gas.

Innervation of the Intestines.

Irritations which strike the mucous membrane first reach the end-fibers emanating from the nervous ganglia in the submucosa, the plexus of Meissner, and are then most probably transferred to the myenteric plexus or plexus of Auerbach. The nature of the nervous mechanism of intestinal peristalsis is still quite obscure.

Experimental investigations concerning the influence of the mesenteric nerves on the peristaltic movements have yielded no certain results. Investigations on the influence of the vagi have yielded the following results: Stimulation of the vagi arouses or increases the peristalsis in the entire small intestine and in the upper half of the colon. The investigations concerning the nature of the fibers contained in the splanchnic nerve have demonstrated that intestinal innervation is a very complicated affair. In the first place, Pflüger discovered that the same portions which are aroused to peristalsis by stimulation of the vagi may be inhibited by stimulation of the splanchnic nerve. The first deduction, then, was that the vagi contained the excitatory fibers and the splanchnics the inhibitory fibers. In addition to this it was later discovered that this inhibitory influence of the splanchnic nerves is a direct one, and that they also contain the vasomotor fibers for the intestinal blood-vessels, for stimulation of the splanchnics causes anemia and their intersection causes hyperemia of the intestine. A clear conception of intestinal innervation was made difficult by the results of Ludwig and Kupffer, von Basch, O. Nasse, S. Mayer. These investigators found, in addition to inhibitory and vasomotor fibers, directly excitatory fibers in the splanchnic, the stimulation of which brought on a limited intestinal peristalsis, and then again Basch and Mayer asserted that stimulation of the vagi could also inhibit peristalsis. This is apparently directly opposed to the results of Pflüger, previously stated.

Some system has been brought into these results by the studies of Ehrmann and von Basch. They suggest that the splanchnic nerve contains the motor fibers for the longitudinal muscles and the inhibitory fibers for the circular muscles. On the other hand, the vagus contains the excitatory fibers for the circular muscles and the inhibitory fibers for the longitudinal muscles. This conception is analogous to one previously formulated by Fellner concerning the innervation of the rectum. He found that the nervi erigentes contained the motor fibers for the longitudinal muscles, and that the hypogastric nerves contained the motor fibers for the circular muscles of the rectum. In addition to the investigators mentioned, a great number of other pathologists and physiologists have interested themselves in this subject. Recently, Steinach claimed to have found motor fibers innervating the intestinal tract which are contained in the posterior sensory spinal roots of lower vertebrates. The motor and inhibitory fibers of the colon and rectum are contained in the sympathetic nerves, which collect and communicate in the inferior mesenteric and hypogastric plexus. Goltz has found a center in the lumbar cord which innervates the lower section of the rectum and is concerned in the act of defecation.

Mechanism of Intestinal Peristalsis.—The movements of the intestine are brought about by contractions of the muscular tunic in the following manner: The outer or longitudinal muscular fibers contract, first producing a dilation of the intestine, and just as they begin to relax, the inner or circular fibers contract, narrowing the lumen and forcing on the contents into the expansion caused by contraction of the longitudinal fibers. Contractions of the muscularis mucosæ can move only the mucous membrane proper and the villi, favoring the exit of the chyle from the interior of the villi (lacteals), and the entrance and exit of the blood into and from the lacteal capillaries, and thereby the resorption of the end-products of intestinal digestion. Peristaltic movements may be caused by stimulating any part of the gastro-intestinal tract. According to Nothnagel, stimulations reaching the intestinal mucosa first irritate the sensory nerve-fibers, and these in turn transfer the stimulus to the motor centers of the intestine. Landois is of the opinion that the myenteric, or Auerbach's, plexus is the sole one innervating the motions of the intestines, and that the submucous, or Meissner's, plexus simply serves for the reception of impulses received through the sensory nerve-fibers of the mucosa and their transmission to Auerbach's plexus. Fleischer inclines to the opinion that Meissner's plexus serves mainly as a motor center for the numerous villi which it supplies with nerve filaments.

In his interesting study on intestinal contraction, F. P. Mall ("Johns Hopkins Hospital Reports," vol. 1, p. 37) employs somewhat different terms for the various peristaltic movements of the intestine. He speaks of the pendulum movements as the "rhythmic contractions," and of the ordinary progressive contraction as the "vermicular action." In addition to this he describes a third form of peristalsis which he terms "irregular rapid wave," and which corresponds to the "Rollbewegungen" of Nothnagel. The rhythmic contractions or pendulum movements may affect a great portion of the whole intestine at the same time, and, according to Mall, are dependent upon the circulation in the bowel. It has been demonstrated that the current in the blood-vessels is controlled to a limited degree by the intestinal peristalsis—that a contraction of the circular muscle may act somewhat as a pump. With each contraction of the circular fibers blood is expelled from the venous plexus of the submucosa, thus increasing the portal pressure. When the same muscles relax again, this blood is prevented from returning by the valves in the intestinal veins. In this manner a rhythmic wave is produced within the superior mesenteric vein. The rhythmic contractions not only regulate the circulation within the intestine, but effect a wave within the venæ portæ, thus aiding the circulation in the liver. Independent rhythmic contaction is by no means rare in involuntary muscles. Laschinger (*l. c.*) demonstrated rhythmic contraction in the veins of a bat's ear when artificial circulation had been carried on for twenty hours after death. Hicks (*l. c.*) showed that the uterus contracts rhythmically once every five, ten, or twenty minutes throughout gestation. During active digestion more blood is required in the intestine, and the rhythmic contractions, by their pumping action, produce a venous pulse which assists the circulation through the liver.

The second form, the slow, regular contraction, always occurs in one direction, and appears to be due to local irritation, because it may be observed working independently in different portions of the intestine at the same time; this is what Mall calls the "normal vermicular contraction." By this motion bodies are propelled about two centimeters in a minute, or, if it is continued uninterruptedly throughout the whole intestine, it would require one hour and thirty minutes to pass over the entire length.

Here we may be permitted to indulge in an interesting bit of speculation concerning the neuromuscular mechanism by which the characteristic vermicular peristalsis is effected. We have already quoted Ehrmann's statement that the longitudinal muscles of the intestine are innervated by the motor fibers of the splanchnic and the inhibitory fibers of the vagus, and that the circular muscles are innervated by the motor fibers of the vagus and the inhibitory fibers of the splanchnic. If one assumes an alternating excitation of the vagus and of the splanchnic, the periodic changes in the contractions of the longitudinal and circular muscles causing the characteristic peristaltic movements could be explained. The excitation of the splanchnic occurring first would effect a contraction of the longitudinal muscles, and simultaneously inhibit the contraction of the circular muscles. Immediately following this the irritation of the vagus would, on the contrary, produce contraction of the circular muscles and inhibit the contraction of the longitudinal muscles. Cash (*l. c.*), in a study of the normal vermicular peristalsis, found that it was due to a rhythmic contraction of the circular muscle above the body, which was being propelled onward. Schillbach (*l. c.*) found that irritation of the intestine by an induced current, as well as by a constant current, produced a local contraction which then ran up the intestine for from twenty to thirty centimeters, and downward for a few centimeters only when the constant current was employed. When the faradic current was used in stimulating, the contraction ran up the intestine for five or six centimeters, and down for only two or three centimeters. Nothnagel (*l. c.*) observed that irritation produced by a crystal of sodium chlorid caused a contraction above the point of irritation. If such a crystal is slowly drawn downward over the peritoneal surface of the intestine, a contraction of the circular muscles will gradually follow it. Foreign bodies within the intestine produce the same vermicular peristalsis, which in this case has the effect of moving the body downward, every new portion of mucosa with which it comes in contact issuing out a new reflex which again moves it downward. The observations which we have quoted demonstrate that an irritation at a given point tends to produce a stronger contraction above the point of irritation.

The nature and mechanism of intestinal peristalsis became still more intelligible after Nothnagel had shown that irritation, when exerted upon one point, caused not only contraction at and above that point, but also dilation below it, and that the dilated portion below the point of irritation gradually telescoped itself over the contracted portion above it, and thus a partial intussusception was caused. These observations make it probable that a circumscribed contraction tends to produce dilation below, and, conversely, circumscribed dilation has a tendency to produce a contraction above. These views have also been suggested by Guérin (*l. c.*) and by von Basch. It, therefore, seems probable that persistent contraction at a point produces dilation below it, and dilation due to the stimulus of intestinal contents or foreign body produces contraction above it. According to these observers and Mall (*l. c.*), there are at least two forces that are active in moving a body through the intestine: first, the body as an irritant causes contraction of the circular muscle about it, which pushes the body downward, and every new portion of the mucosa thus irritated causes a new contraction above the point of irritation; second, simultaneously an active dilation below the constriction produces an aspirating force having a tendency to suck the body downward.

The irregular or rapid wave, which corresponds to the "Rollbewegungen," or, more likely, to the pathologically increased peristalsis, is best seen in the intestine of animals just killed. According to Mall (*l. c.*, p. 69), it passes throughout the whole length of the intestine in about a minute. When two passing waves meet, they check each other. The fact that Mall could not demonstrate this peristaltic wave in the living animal except under certain conditions, and that it progressed at so surprisingly rapid a rate, suggests that this form of peristalsis is distinctly pathological. When the intestine is very anemic or hyperemic, it becomes extremely irritable. This is due in both cases to want of oxygen. Bokai could produce increase in the peristalsis by introducing CO_2 into the intestinal canal, but he could allay the peristaltic excitement again by introducing oxygen. He also found that a very irritable state was brought about by the gases and organic acids of decomposition. It is, therefore, rational to conclude that the irregular rapid contractions thus spoken of are the means of speedily ridding the intestine of irritating products of decomposition. They do not occur during normal digestion.

Influence of Disturbances of Circulation on the Peristalsis.

Every interruption in the movement of the blood current in the intestine causing either anemia or venous hyperemia produces, in the beginning of the disturbance, an increase in the rate of the peristalsis. Salvioli (*l. c.*) demonstrated, in excised pieces of the intestine through which he carried on an artificial circulation, that interruption of the blood current caused contraction of the intestinal musculature and peristaltic waves. When arterial blood rich in oxygen was allowed to flow into the portion of the intestine, it resumed its state of rest. The irritating condition, therefore, in these circulatory disturbances is deficiency in oxygen and accumulation of carbon dioxid.

Very slight circulatory disturbances may be followed by an increased peristalsis. The increased peristalsis observed in moribund patients or in the state of marasmus is also attributable to beginning disturbances in the circulation of the intestinal vessels and alteration of the gases of the blood. When the blood of another species was transfused into an animal, Landois observed stases and occlusions of the intestinal vessels, which resulted in a stormy peristalsis accompanied by loud rumbling and borborygmi in the intestines and involuntary evacuations. When circulatory disturbances persist for a long time, they finally produce exhaustion of the muscular layers and complete arrest of the peristalsis. Any irritation, such as inflammation of the peritoneum or of the mucosa or submucosa, persisting for a long time may cause intestinal paralysis. It has been observed, in patients affected with valvular troubles, that the intestinal peristalsis may be normal, increased, or diminished, according as the compensation is complete, partial, or incomplete. An analogy to these studies may be found in the observation that, when arterial blood is introduced in the vessel of a relaxed intestine, it first cau [illegible] sed and then a normal peristalsis. If circulatory disturbances cease spontaneously, or if they can be relieved in patients by administration of digitalis, strophanthus, or strychnin, and the venous intestinal hyperemia can be corrected, at first abnormally active peristaltic movements result, and later on they become normal. It may happen that the weakened and relaxed intestinal walls are so excessively expanded by the stagnating contents and abundant formation of gases that the contractility of the musculature becomes seriously impaired.

Pathological Intestinal Peristalsis.

The manifold irritations which may strike the intestinal mucosa are most probably first transferred from the sensory end fibrils to the submucous plexus, and from this to the myenteric plexus, which is the independent motor center of the intestine. The latter, also called the plexus of Auerbach, may have its activity stimulated and inhibited not only by impulses emanating from the intestinal mucosa, but also by such emanating from the blood, the peritoneum, the brain, the spinal cord, as well as from other organs, particularly the stomach. The exact route by which these innervations travel and the rôle of intervention which the vagus and the splanchnic play in their transference are unknown. The changes in form and position of the intestine and the consequent movements of the contents will proceed in a normal manner when the following conditions are fulfilled: (1) The digestive stimulation of the intestinal contents must be maintained at a certain level; a certain amount of food irritation is necessary for healthy peristalsis. When this is excessive or insufficient, the peristalsis becomes augmented or diminished in proportion. (2) The irritability of the motor or sensory nerves of the intestine must be normal. (3) The contractility of the intestinal musculature must be normal. (4) The rate of the blood flow in the intestinal vessels, the composition of the blood, particularly its gaseous constituents, must be normal. (5) Stronger excitatory or inhibitory impulses from other localities in the organism must not reach the intestinal motor centers.

In case of abnormal intestinal peristalsis, if it can be ascertained which one of these five conditions is altered and in what manner, the necessary consequences in the intestinal movements may be deduced with some certainty. Some morbid alterations, when they have reached a very advanced degree, may in the beginning cause an increase in the peristaltic movements, but after they have existed for a longer time they may produce a weakening and even a complete arrest of the peristalsis; this is found to be the case in venous hyperemia, in duodenitis, jejunitis, and ileitis. Disturbances of the peristalsis will ensue when any one of the five conditions mentioned is not normal. When several of the conditions are abnormal,—for instance, if we have simultaneously increased irritation of the mucosa and increased sensibility of the sensory and motor nerves,—we will have a very active peristalsis resulting in case the musculature is intact. If, in addition to this, a venous hyperemia is added, the altered conditions all act in the same direction, and the result is due to a summation of irritations which is capable of effecting a stormy peristalsis and even involuntary evacuations. When there is an increased excitability of the intestinal nerves, very weak irritations may cause normal or increased peristaltic movements which would not be produced under normal conditions. The motor center of the intestine may be in a state of increased excitation, it may be inhibited, or it may be abnormally excited and inhibited at the same time. The influence of one kind of disturbance upon the peristalsis may be totally counterbalanced by an antagonistic influence. When excessive demands are made upon the working capacity of a musculature by strong irritations which are persistently repeated, a state of exhaustion and paralysis of the intestine results; this may be observed in the behavior of the intestinal movements associated with stenoses. When the obstruction is moderate, the musculature in the suprastenotic portions of intestine becomes hypertrophied, in order to surmount the difficulty of the passage; but when the obstacle has become insurmountable and the most intense muscular efforts of the intestine can effect no passage through the stenosis, we finally observe paralysis of the portion involved; this paralysis may be brought on more rapidly after the administration of cathartics.

Nothnagel classifies the pathological intestinal peristalses under three headings: (1) Abnormally increased peristalsis; (2) tonic or tetanic contraction; (3) antiperistalsis.

1. Increased peristalsis may occur under the following circumstances: (*a*) Abnormal fermentations and putrefaction of the intestinal contents, causing increased acid reaction and the formation of toxins and gases; (*b*) the influence of extreme heat or cold or chemical intestinal irritants; (*c*) the introduction of purgatives; (*d*) under the influences of psychic and emotional impressions; (*e*) in neurasthenics; (*f*) in acute inflammatory conditions of the intestine and (*g*) in enterostenosis.

The rolling intestinal movements, which we have already described under the normal peristalsis, and which Mall designates as the "irregular rapid wave," form the transition from the normal to the abnormal peristalsis. Several types of pathological movements have been observed occurring simultaneously. A combination of normal and abnormal movements has also been observed. Thus, Mall (*l. c.*, p. 73) states that he has frequently noticed the rapid waves pass over the same loop of intestine in a rhythmic manner. This would be a combination of Nothnagel's "gesteigerte peristaltic" with "pendelnde Bewegungen"—an abnormal with a normal movement. When the intestinal mucosa is in a state of inflammation from a chemical agent, one can observe very violent contractions in the inflamed district, even when this is entirely empty. Nothnagel compares this result to an uninterrupted burrowing tremor, in which he observed rhythmic pendulum movements and circular constrictions occurring simultaneously or alternately. According to Nothnagel, the important condition constituting abnormal peristalsis consists of the increased rapidity with which the contents are moved downward. It is difficult to determine to what extent the different forms of intestinal contraction may blend into one another.

2. *The tetanic contraction* presents two different clinical pictures: (*a*) the tetanic contraction occurring with an empty, and (*b*) with a filled, intestine. The tetanic contraction of an empty intestinal district converts the tube into a pale, solid string. If it has extended over a great number of intestinal loops, it causes a concavity of the abdomen, such as is seen in cerebral meningitis and lead colic. The second form of tetanic contraction occurring in a loop filled with intestinal contents is frequently seen in intestinal stenoses. The loop, which is excessively filled with liquid and gaseous contents and which felt soft and elastic to the touch, suddenly becomes very resistant and is converted into an almost rigid tube. In a short time it returns to its previous elastic state. Mall concludes

that the classical vermicular wave ("fortschreitende peristaltische Bewegung") is pathological rather than physiological, because it results from the action of a chemical substance—CO_2 or skatol; that it usually works in one way in order to rid the intestine of improper products of digestion and decomposition. It is evident that if this view is applied too strictly, we may have to conclude that there are no normal contractions of the intestine at all, because they are in the end, all of them, produced by the intestinal contents, frequently by the products of digestion. Then, also, it is not at all certain that CO_2 and skatol are abnormal products of digestion. In our opinion the classical vermicular wave is not pathological, because it is never accompanied by pain, no matter how active it may be, while, on the other hand, the tonic contraction is connected with intense colicky pain. The pains which occur with enteritis, in which, as is well known, very active vermicular peristalsis occurs, are not assigned to this form of movement by Nothnagel, but to the simultaneously occurring tetanic contractions, the irritability of the nerves, and the inflamed condition of the mucosa.

3. Antiperistalsis.—Experimenters have now almost unanimously come to the conclusion that normal peristaltic action works in one direction only. The clearest demonstration of this fact has been furnished by Mall ("Reversal of the Intestine," "Johns Hopkins Hospital Reports," vol. 1, p. 93). To give this a thorough test, Dr. Wm. S. Halsted reversed several loops in dogs for this investigator. A loop about thirty centimeters long was simply turned around, and sutured in such a manner that the food, in passing from the stomach to the rectum, had to pass through a portion of the intestine in the reversed direction. The animals lived over a month, and seemed to die of starvation. In both cases the reversed portion was enormously dilated about the suture lying nearest to the stomach, and the mucous membrance of that portion was converted into fibrous tissues. That part of the intestine was clogged with food and such materials as had been given the animals to eat during the experiment—straw and glass balls among other things. It was very evident that although the intestine was reversed, it still persisted in working in its old way, which was now turned toward the mouth, and had retained all the solid particles in the neighborhood of the upper suture; the lower sutures were fully healed and not distended. Antiperistaltic action occurs only under certain abnormal conditions, which, according to Nothnagel, consist in the introduction of either strongly irritating chemical substances—for instance, strong solution of sulphate of copper—or chlorid of sodium and the like. Even this abnormal irritation produces a typical peristaltic movement simultaneously with the antiperistaltic movement, but only then when the irritant substances are introduced at an unphysiological entry. In man the rectum must be regarded as such. When these irritating substances are introduced into the stomach and reach the intestine, they create only regular downward peristaltic movements.

It was formerly believed that antiperistaltic action took place in volvulus, intussusception, invagination, and axial twists of the intestine, causing sudden stenosis, because in these conditions vomiting of fecal matter had been observed, which could not be explained at that time except by assuming an antiperistaltic action of the bowel. Nothnagel has shown that in stenoses experimentally produced, a very active peristalsis first occurs, together with an increased secretion of liquids, causing a distention of the section of intestine in front of the stenosis. After the dilation of the intestine has become considerable, it contracts strongly; by this the contents are forced into the adjoining superior portion of the bowel. This procedure may progress from a limited distance upward. Nothnagel does not believe that this backward contraction can effect the propagation of intestinal contents into the stomach in patients affected with volvulus, because the portion of the bowel immediately in front of the stenosis is put into a state of paralysis from excessive dilation in a very few hours. He attributes the stercoraceous vomiting which occurs in volvulus to the exertion of the strong abdominal pressure on the highly distended paralytic intestinal portions occurring in front of the stenosis. This view is in agreement with that of Van Swieten. When the stomach is perfectly empty, the pylorus is only lightly closed, and the entrance of intestinal matter thereby facilitated. Nothnagel produced invagination experimentally by powerful faradization of a short piece of the intestine, and Exner has given a theory of the mechanism of intestinal invagination, which we shall consider in a special chapter.

In a very interesting series of experiments Grützner ("Deutsch.

med. Wochenschr.," 1894, No. 48) demonstrated that under certain conditions starch granules, particles of charcoal, finely cut horsehair, sawdust, etc., that were impregnated with normal salt solution and injected into the rectum of rabbits, guinea-pigs, and rats were found six hours later all along the upper part of the small intestine, even in the stomach. When suspensions of these particles were made in distilled water, dilute HCl, or potassium chlorid solution instead of physiological salt solution, the particles did not descend into the digestive tract. (For a critical review and experiments on Grützner's results see "Diseases of the Stomach," by J. C. Hemmeter, second edition, pp. 211–214.)

The ascent of food particles into the stomach from the rectum, as observed by Grützner, is invisible to the eye, but the antiperistaltic movement produced by a salt crystal in the experiment of Nothnagel is very plainly visible. The antiperistaltic motion of Grützner we conceive to be only a very feeble marginal ascending movement, effected by surface contact of particles with the epithelium, which is moved by the muscularis mucosæ. It is doubtful whether this invisible upward movement is capable of propelling food masses, because a central downward current of large masses of contents occurs simultaneously with it. Grützner assumes that nutritive enemata are moved upward through the entire small intestine and so become digested and absorbed; even Riegel (*l. c.*, p. 245) and Fleischer (*l. c.*, p. 1045) are satisfied with this interpretation. In my experience the bulk of nutritive enemata does not ascend out of the large intestine. Very often, when such enemata are given three times daily, a large part of the morning enema is washed out of the colon by the cleansing injection by which the new enemata are preceded. It appears that Riegel and Fleischer regard the ascent of particles observed by Grützner as an actual antiperistalsis, equally powerful and capable of moving large food masses as the regular downward peristaltic movement; this is, in our opinion, not the case. In accordance with Christomanos, we could never demonstrate a visible antiperistalsis in our repetition of Grützner's experiments.

From what I have said in a preceding chapter it is probable that egg-albumen, fats, and milk may be absorbed from the colon as such without being digested. It is also quite probable that a proteolytic ferment working in an alkaline medium occurs in the contents of the colon, and that the digestion of food enemata may be due to this agent. Busch ("Virchow's Archiv," Bd. XIV) found that food injected into the rectum appeared hours, sometimes days, after, from an intestinal fistula; it has also been noticed in rare cases of great emaciation that food substances have been vomited after having been injected into the rectum. These are observations concerning decidedly pathological conditions. Busch also observed that solution of coagulated albumin took place in the intestine in the absence of gastric or duodenal secretions: the patient had received an abdominal injury, having been horned by a steer, so that the gastric juice, together with the bile, pancreatic juice, and succus entericus, ran out through a fistula.

It has already been stated that the chyme traverses the small intestine in from two and one-half to five hours, and that the much shorter distance from the cecum to the rectum requires twelve or more hours. This rapid movement through the small intestine prevents the increase of putrefactive bacteria and their decomposing influence on albuminous substances; the slow peristalsis of the colon, however, and the stagnation of the contents caused thereby, favor putrefaction. This decomposition would be still greater in the colon but for the fact that six-sevenths of the total amount of proteid matter is digested in the small intestine, and only one-seventh left for the large intestine.

Defecation.

The contents of the colon become more and more condensed by the absorption of water, until they are converted into a mass of some consistency, generally filling the entire lumen of the large intestine. When it has reached the lower section of the rectum, this column of excrement causes irritation of the sensory nerves and subsequent active peristaltic movements of the lower part of the gut. The act of expelling the feces from the rectum is the result of an interesting physiological mechanism. It is effected by the combined forces of the peristalsis of the lower bowel, contraction of the abdominal muscles and energetic descent of the diaphragm, which effect a very rapid descent of the fecal mass. Landois has called attention to an additional factor favoring the extrusion—the passive congestion which occurs in the intestinal vessels as a consequence of prolonged and persistent abdominal pressure before and during defecation. From what we have learned above, the overfilling of the intestinal vessels with venous blood causes active peristalsis.

The inhibitory center for the sphincter ani muscles, which has been referred to the optic thalami (the fibers passing through the cerebral peduncles and medulla to the lumbar cord), produces a temporary relaxation of the sphincters. A tonic contraction of the same, which might reflexly be caused by strong irritation of the sensory rectal nerves, would make defecation impossible. While the inhibitory center produces a relaxation of the sphincter muscles, the levator ani contracts, thereby stripping the mucous membrane of the rectum over the descending column of excrement and preventing a prolapse. We shall speak of the pathology of defecation under the head of Constipation.

The Influence of Medicinal Agents on the Peristalsis.*

Among the chemical stimulants of the mucosa during the normal digestion of food-stuffs in the intestines we must class the HCl of the stomach-contents, the bile, and the alkaline secretion of the pancreas and intestinal glands. To this group are to be added the organic acids which arise from fermentation of carbohydrates. Prominent among these are butyric, lactic, acetic, formic, and caproic acids. It was formerly believed, concerning the action of the neutral saline purgatives, that their effect was due to large quantities of water which were secreted into the intestine from the blood-vessels, and which kept the contents in a liquid condition. This property was ascribed to the hygroscopic power of the saline. From the experiments of Rodziejewsky and Thiry it is evident, however, that the method of action of all purgatives is dependent upon a stronger irritation of the motor center of the intestine, and a consequent powerful excitation of the peristalsis. Concentration and thickening of the contents impede the normal progress of the feces. The neutral salts act also by stimulating the peristalsis, but it is probable that the hygroscopic property, by retaining considerable water in the intestine, aids in the purgative effect. It is not necessary for drastic remedies to come into actual contact with the intestinal mucosa in order to produce their effect: they may act from the stomach or even from the blood. Mild laxative remedies act directly on the intestinal mucosa; they have no such powerful effect from the stomach and blood as is observed with the drastics.

---

* See also chapter XIII, Important Medicinal Agents.

It has been repeatedly observed that putrefactive substances injected into the circulation cause a violent persistalsis by influence of the toxins on the motor center of the intestine. W. D. Booker ("Transactions of the Ninth International Medical Congress," vol. III), who has isolated many of the bacteria of the intestines occurring in enteritis of children, found that some of the organisms produce substances which, when injected into the intestine, produce very powerful contractions. The animals experimented upon were young rabbits whose abdominal cavities were opened under normal saline solution. The fluid containing the toxic substance was injected with a hypodermic syringe into the duodenum. In a short time the duodenum began to contract above the point of injection; these contractions gradually extended over the point of injection and forced the fluid downward. The contraction would begin above and run downward, then, after a short interval, repeat itself again and again, making a rhythmic peristalsis. As a rule, the contractions were not in advance of the fluid propelled. Potassium salts always cause a local contraction, while sodium salts always cause a contraction above the points of irritation (Nothnagel, "Virchow's Archiv," Bd. LXXXIII). According to Bardeleben ("Virchow's Archiv," Bd. LXXXIX), potassium and sodium salts act alike on the human intestines. The contraction caused by potassium salts results very promptly in about one-half or one second after the contact, and lasts for ten minutes. The contraction due to a contact with sodium salts is not limited to the spot touched, but may extend ten centimeters above it, always toward the pylorus. It occurs much later than the contraction caused by potassium salts—namely, two to eight seconds after contact. It begins at a point from two to four millimeters above the spot touched, and lasts from five to thirty seconds. The circular constriction which is caused thereby is frequently repeated and generally followed by a vivid peristalsis. Nothnagel concludes that the sodium salts act principally upon the nerves of the intestine, and that particularly the ascending contraction, which is constantly observed, is transmitted by the nervous elements and not to be regarded as a muscular effect, while the potassium salts effect a stronger contraction of the unstriated muscular fibers than the sodium salts.

The Effect of the Intestinal Gases.—Carbonic acid gas (CO_2), hydrogen sulphid (H_2S), and marsh-gas (CH_4) are effective excitants of peristalsis, while oxygen has a quieting effect on the bowel movements. We have already referred to Bokai's interesting observation that energetic intestinal contraction will follow when an animal is suffocating, and these may be arrested again by allowing the animal to breathe freely or by passing oxygen into the lumen of the intestine. The CO_2 had the same effect upon the intestine when it was introduced into the lumen as when it was introduced through the blood-vessels.

Effect of Drugs, Chemicals, and Toxins.—Phenol and skatol produce powerful contractions of the intestine, while indol seems to be without effect. Belladonna and atropin reduce the irritability of the plexus of Auerbach, thereby causing reduction, and in large doses even arrest of the peristalsis. Opium and morphin in very small doses excite the inhibitory fibers of the splanchnic, but in large doses they paralyze the same. Therefore, like belladonna, these drugs favor constipation. Caffein, muscarin, and nicotin excite the motor apparatus of the intestine. They therefore favor evacuations, but nicotin in larger quantities produces intestinal spasm. Lead salts also produce intestinal spasm; this has been observed in cases of lead-poisoning, and is due to a simultaneous tonic contraction of the longitudinal and circular muscular fibers. This spasm is relieved by morphin and opium and is increased by purgatives.

CHAPTER V.

UTILIZATION OF FOOD IN THE STOMACH AND INTESTINES OF THE HEALTHY MAN.

The utilization of food in the intestines of the healthy man is dependent on two conditions: first, on the kind, form (preparation), composition, and amount of the same, which, in view of the great number of the food-stuffs in question, varies greatly; and, secondly, on the digestive capacity of the intestines and of the organs concerned in digestion.

Kinds of Food.

With regard to their derivation, foods may be divided into two great groups—these are the animal and the vegetable foods. To the latter belong also the relishes and spices, which, although they usually contain only very small amounts of albumin, fat, or carbohydrates, yet play an important rôle in nourishment, because they give the food its agreeable taste and are therefore indispensable. By means of numerous careful experiments in nourishment and utilization of food made by Rubner, and also by Atwater, Praussnitz, and Uffelmann, it has been determined that most vegetables,—that is, the food-stuffs contained in them, and especially the albuminates,—are not thoroughly utilized in the digestive canal as are the animal foods, in which category eggs, milk, cheese, and butter are to be placed. While 97 per cent. of the albumin of meat and eggs and 90 per cent. of the albumin of milk are absorbed in the intestines, only 68 per cent. of the albumin of rye bread and of potatoes, 72 per cent. of the albumin of soaked peas, and 78 per cent. of the albumin of wheat bread are absorbed. On the other hand, the carbohydrates of the latter are thoroughly utilized. Besides, the utilization of meat might vary according to the age of the animals that supply it. The tender flesh of young animals, which is poor in connective tissue, is certainly always utilized more thoroughly than the flesh of older animals, which is rich in connective tissue. The meat of young pigeons, chickens, partridges, hares, calves, pigs, kids, and also of fish, lobsters, crabs, oysters, and mussels, is eaten, as a rule, shortly after the death of the animals. The flesh of steers, oxen, cows, sheep, black game (boars, bears, and badgers), old hares, partridges, roes, deer, etc., is not eaten until some time after the death of the animals—at any rate, not before the death rigor has passed away. After longer keeping (hanging of the flesh of older animals) large amounts of sarcolactic acid are formed in the muscles, which reacts on the connective tissue and muscular fibers, softening and breaking them up, and thus promoting the digestibility and utilization in the intestines. By putting meat in sour milk, which is rich in lactic acid and also in vinegar (acetic acid, which acts similarly to lactic acid), this process may be hastened. Those fats which are fluid at the temperature of the body are absorbed almost completely in the intestines—according to Voit and Rōhmann 98 to 99 per cent., while only 90 per cent. of those fats are absorbed which have a semisolid consistency at the temperature of the body (J. Munk, Fr. Müller), and only 11 to 13 per cent. (E. Voit) of those which are solid at the temperature of the body (see W. O. Atwater, " Methods and Results of Investigations on the Chemistry and Economy of Food," Bulletin No. 21, Office of Experiment Stations, United States Department of Agriculture).

Composition of the Food.

A diet consisting of albumin, fats, and carbohydrates, in which the proportion of the albumin to that of the fats and the carbohydrates is as 1 is to 4, is best assimilated in the intestines, according to the experiments that have been made up to the present time. Every excessive favoring of one of these three food-stuffs at the expense of the other must be avoided, because their utilization in the stomach and intestines is unfavorably influenced thereby. Thus, for instance, if the food contains an abnormally high percentage of carbohydrates, 20 per cent. of the albumin introduced at the same time may appear in the excrement unutilized.

Amount of Food.

The amount of nutritive substances that the normal gastro-intestinal tract is capable of absorbing is, at the most, only 300 gm. fats, 400 gm. albumin, and 550 gm. carbohydrates in twenty-four hours; but by the introduction of such amounts great demands are made on the intestines and the stomach, and there are probably very few men who would not, after the introduction of such great amounts of fats, albumin, or carbohydrates in their food, pass off a part of the same unutilized in the feces. So large a quantity of any one of the food-stuffs is assimilated completely or nearly completely in the intestines only when an increase in one of these substances is accompanied by a proportionate diminution in the quantity of the others.

Preparation of the Food.

The digestibility of vegetable and animal foods may be materially increased by a judicious preparation of the food by means of roasting, boiling, baking; furthermore, by beating the meat, cutting it into pieces, or grinding or scraping. The utilization of animal foods is not in all cases improved by boiling, roasting, or smoking, certain kinds of meat, especially beef and probably also ham, being better assimilated when in a rare condition. The frequent use of raw meat is not advisable, however, because raw meat soon excites repugnance, and, on the other hand, because there is danger of the transmission of animal parasites unless the meat is inspected microscopically (see Dietetic Kitchen, Hemmeter's "Diseases of the Stomach," second edition, p. 228).

Behavior of the Healthy Intestine During Digestion.

The digestive capacity of the intestines is naturally not the same in all people; the working power of its secretions differs just as does the capacity of the glands which produce them. There are individual fluctuations, which can often be verified in practice. While some individuals assimilate entirely or nearly entirely very great amounts of food, others pass off parts of the albuminates, carbohydrates, or fats unutilized with the feces if they eat the identical amounts. Sometimes all the members of a family are distinguished by a marked digestive power of the stomach and intestines; indeed, this may be inherited through several generations. From personal experience in private practice I can state that in some families all the members complain of infirmity of their digestive organs, and that after heavy meals the feces contain large amounts of undigested particles of food.

The utilization of the various food-stuffs—albuminates, fats, and carbohydrates—may be quite unequal in different persons, so that many people may thoroughly assimilate very large amounts of fats that are liquid at the temperature of the body, or large amounts of carbohydrates, and yet, on the contrary, assimilate the other two food-stuffs incompletely. Undoubtedly much depends upon habit. On this account Japanese and Irishmen who from youth up are accustomed to live quite extensively on rice and potatoes, respectively, can assimilate 650 gm. of these, whereas most other persons who from childhood have consumed greater quantities of albumin and fats besides the carbohydrates can at most assimilate and absorb only from 500 to 550 gm. of carbohydrates. When the diet is changed,—for instance, when one passes over from a diet consisting chiefly of meat with but few vegetables to one consisting largely of vegetables with but little meat, or vice versâ,—the new nourisment will probably at first be incompletely assimilated, and continue so, indeed, until the secretory apparatus of the stomach and of the intestines has grown accustomed to the stronger or weaker (mechanical and chemical) stimulation, as the case may be. Also, in the case of convalescents recovering from severe fevers (typhoid), who have for many weeks taken but scanty liquid or pulpy nourishment, when larger amounts of food which is not so easily digested are introduced, the food is certainly at first less thoroughly absorbed than before the sickness, and probably a shorter or longer time will elapse in the various cases before the capacity of the digestive organs has again reached its previous condition.

Utilization of Food in the Intestines under Abnormal Conditions.

When the flow of the bile into the intestines is completely stopped, the digestion and absorption of fats and also of albuminates suffer; especially so if the food contains much fat, because the undigested fat envelops a part of the albumin of the food and thus hinders its transformation and absorption into the lacteals. If the secretion of the pancreatic juice into the intestines ceases completely from any reason, all three food-stuffs are very probably less completely utilized than normally. The same is transiently the case during extended acute catarrh of the large and of the small intestine; permanent defective utilization of food is produced in severe amyloid degeneration, and also in atrophy of the intestinal mucosa and muscularis. The fact that those people who suffer from stomach diseases often assimilate the food more incompletely in the intestines also, need not surprise us when we consider the close interrelations existing between the two portions of the alimentary canal. If, for instance, in patients suffering from dilation of the stomach, chronic gastritis, or cancer, serious decomposition and decay take place in the contents of the stomach as a result of long-continued stagnation and of the absence of free HCl, these contents, when passed over into the intestine, may irritate the mucosa and its nervous motor end-organs so much that the chyme is carried down too rapidly, and a part of the nourishment contained therein is not digested and absorbed, and is passed off unutilized with the feces. In those patients suffering from nervous gastric anacidity, the albuminates, carbohydrates, and fats of the food are utilized less completely than in the case of normal secretion of gastric juice. For when there is no free HCl in the gastric contents, the putrefaction of the chyme may start already in the smaller intestine, and the efficacy of the intestinal ferments, as is well known, may be completely destroyed, after a short time, by bacteria and toxins.

The interesting physiological experiments of Pawlow (*l. c.*) have already been referred to, but I would repeat here that, according to this investigator, the HCl of the gastric juice is the normal stimulation to the secretion of the pancreas—in the absence of HCl, I have, as a rule, generally found the pancreatic digestion less active than normally. This occurs also in all those diseased conditions of other organs which either cause (directly or indirectly) an extreme venous passive congestion in the digestive organs, for instance, in affections of the heart, the respiratory organs, the kidneys, the vessels, the liver, etc., which react unfavorably on the motility and absorption, as well as on the secretion of the intestinal juices, or which, through the medium of the nervous system, cause a great increase of intestinal peristalsis, cutting short the time of digestion of the chyme in the intestines. Atony and even intestinal spasm may occasionally be observed in meningitis and lead intoxication. All these diseased conditions may affect the utilization of the food in the intestines more or less unfavorably. A judicious diet adapted to the changed conditions can be selected only after a thorough knowledge and careful consideration of the pathological changes in the various diseases.

Digestibility of the Various Foods.

The word "digestibility," in spite of its frequent use, is variously defined, and many different processes and conditions are included under the term. By some the digestibility of food is identified erroneously with its utilization in the stomach and intestines. Although they are related terms, yet digestibility and utilization are not completely synonymous. For instance, a very abundant, difficultly digested nourishment may be splendidly assimilated in the stomach and intestines if its preparation is correct, and if the digestive organs of the person in question are vigorous. Nourishment, consisting of various foods, of which each one by itself is easily digested, may be but poorly assimilated if the combination is injudicious and the amount is excessive in proportion to the capacity of the stomach and of the intestines. The manner in which foods agree with individuals—*i. e.*, the subjective condition during digestion—is taken by some as an infallible criterion for the digestibility of the same; and those foods are called easily digested which cause no complaint when consumed in large amounts, while those after the consumption of which disagreeable sensations are experienced in the abdomen, are referred to as difficult of digestion. Agreeableness, however, is no reliable criterion, for whereas complaints may not set in after eating foods which are recognized as hard to digest and which are poorly assimilated in the stomach and intestines, the same complaints may often occur after eating food which is thoroughly assimilated; indeed, a form of food which has been repeatedly consumed and splendidly digested and which has never disturbed the subjective well-being may, on another occasion, cause extreme discomfort in the same person, because the irritability of the digestive sensory nerves has been, in the mean while, abnormally increased as a result, perhaps, of excessive mental work or for some other reason. Being accustomed to a certain diet may also contribute to the greater agreeableness of the same, even if it is difficult to digest. A negro consuming five kilos of sweet potatoes in a day may feel perfectly well, although he may be badly nourished and although this diet is entirely faulty and hard to digest and would cause the greatest discomfort to a man accustomed to a diet consisting principally of meat foods. It has been suggested that the digestibility of the various foods could be determined by their longer or shorter stay in the stomach, and that those foods which "the stomach got through with most quickly" are to be considered more easily digestible; those which require a longer gastric digestion, therefore, more difficult of digestion. This opinion, which even now has many adherents, can no longer be maintained, since it is known that the larger part of the food-stuffs is not digested and absorbed in the stomach, but only in the intestines, and that larger portions of fat may pass over into the intestines, although the same is not digested and absorbed in the stomach at all, and that the same may be the case in regard to foods which are entirely indigestible in the stomach and intestines, which foods have been consumed together with easily digestible foods.

The digestibility of various foods and its relation to the rate of evacuation from the stomach are dependent upon a number of factors, some of which are still imperfectly understood. Prominent among these are, first, the amount of HCl required in the proteolysis of any particular food, for this determines the time of the appearance of free HCl in the gastric chyme, and, secondly, the condition of the neuromuscular apparatus of the stomach.

The appearance of free HCl is a stimulus to peristalsis, and hence the less HCl a food requires for its saturation, the sooner it will be rendered soluble or evacuated into the duodenum. For instance, if a healthy individual consumes, on different days, exactly the same amount of either calf's-brain, sweetbreads, veal, beef, pork, and mutton, as a rule the stomach will be found empty sooner when the first two have been eaten than when the others have been introduced, and a strong free HCl reaction can usually be observed sooner in the drawn contents of the stomach when the first two have been eaten than in case of the latter. Since it has also been determined, by many thorough investigations of Fleischer, that sweetbreads and calf's-brain unite with much less HCl, and are correspondingly much poorer in albumin than the others (thus, according to this author, 100 gm. calf's-brain unite with only 0.65 of HCl; 100 gm. of sweetbreads, with 0.9 of HCl; whereas the other meats referred to require more than double or triple the amount of HCl), it is comprehensible that the stay of calf's-brain or sweetbreads in the stomach must naturally be shorter. Experiments on healthy persons and animals have taught us that in the second period of stomach digestion, during which the chyme contains free HCl, its quantity decreases much more rapidly than in the first period, when free HCl is absent, and that in the second period the intervals between the passage of the single portions of the gastric chyme into the intestines are much shorter than in the first. If the chyme contains large amounts of free HCl, not only is a part of the albumin of the food transformed into acid albumin, propeptone, and peptone, which is at once absorbed, but energetic contractions of the gastric musculature are produced by the strong chemical irritation of the gastric mucosa, and thereby the digestion and assimilation of the stomach-contents, as well as their evacuation into the intestines, are materially aided. In case larger amounts of carbohydrates are introduced with the meat foods, the amount of chyme decreases more slowly in the second period of stomach digestion than when the diet consists of meat alone, because the conversion of starch by means of the saliva is inhibited when free HCl (0.12 per cent.) appears. The amylaceous foods, therefore, after this degree of acidity has been reached, can no longer be converted into erythrodextrin, maltose, and dextrose, and must be evacuated into the duodenum in the form of insoluble starches.

Rapid evacuation of certain foods from the stomach—as, for instance, calf's-brain or sweetbreads—does not always indicate that these are more easily digested than others which are retained much longer in the stomach. Under certain conditions there may be evidences of arrested digestion when the process has actually been very active. In the experiments on artificial digestion by Fleischer, for example, equal amounts of calf's-brain, sweetbreads, tender ham, veal, beef, pork, and mutton, each chopped fine and cooked, were subjected to the same conditions of experimental digestion at body-temperature after water had been added. First, they were subjected to the action of HCl and pepsin, and, after the acid had been neutralized and made slightly alkaline, they were subjected to the action of pancreatin. The digestive period was in all about from eighteen to twenty hours. These experiments apparently showed that, contrary to the previous theory, calf's-brain, sweetbreads, and tender ham, which have so often been claimed as easily digested, are less completely digested and utilized in the stomach and intestines than the other kinds of foods. After the completion of the experiment, the digestive mixture of calf's-brain and ham still showed a very considerable undigested residue. In the digestive mixture of the ham there were numerous apparently entirely unchanged pieces of meat which were not decomposed nor even attacked by dilute caustic alkalis; a much larger part of the sweetbreads was digested, but yet a considerable part was not digested, while the other kinds of meat were decomposed and therefore digested up to a very small fraction. Since, however, the results of Fleischer's experiments in glass vessels can not be directly applied to the normal human digestion in the stomach and intestines, and since they need confirmation, it is very desirable that numerous thorough experiments on healthy persons under approximately like conditions be made, for the purpose of determining the digestibility of the most important meat foods. Much depends on the manner in which foods are combined. Thus, fats combined with carbohydrates (butter or gravies with potatoes, butter and rice, bread and butter) are much better utilized than when fats are ingested together with proteids (meats fried in fats or fried oysters), because, in the first place, the two foods, fats and starches, act synergistically, for fats depress the gastric secretion and hence favor the action of ptyalin; but for this same reason meats eaten with fats would not meet with sufficient acid gastric juice to undergo sufficient proteolysis in the stomach. Many vegetable foods, if prepared properly or combined properly, are utilized better in the stomach and intestines than they would otherwise be, as Rubner's experiments have shown.

Proteolysis at First Rapid, May Be Arrested Later by Its Own Products.

It is a well-confirmed observation that the action of ferments is inhibited and may be arrested entirely in the presence of an excess of their own products. For example, ptyalin will cease to effect amylolysis in a glass vessel when the presence of maltose and dextrose has reached a certain percentage. The yeast ferment, zymase, will cease to convert maltose and glucose into alcohol after a certain amount of alcohol has been formed. Numerous other examples of this phenomenon could be cited. We must, therefore, expect that in the action of enzymes the continuous and complete decomposition of the original substance on which the enzyme works can not take place unless the ultimate product formed is removed as fast as it appears. If this is not done, the action will go on rapidly at first, but gradually more slowly, until the equilibrium point is reached, when the action should cease. Tamman has shown * that the action of emulsin on amygdalin is inhibited by the accumulation of the products of decomposition. Brown and Morris have shown that it is not possible to hydrolyze more than 80 per cent. of starch by means of diastase, generally nearly 20 per cent. of dextrin remaining. But Lea has found that the hydrolysis can be completed if the sugar is removed as fast as it is formed.

In an extensive series of similar quantitative digestion experiments, conducted with every regard to physiological and chemical detail, I have been convinced that the same conditions prevail with regard to the proteolysis of the albumin molecule under the action of the pepsin and HCl and under that of trypsin. Pepsin and trypsin do not effect the conversion of proteid into peptone and amids by a single operation, but by a succession of stages, each characterized by the appearance of definite intermediate bodies, which have been isolated and studied by Kühne and Chittenden. I have already described pepsin proteolysis and the views of authorities on the immediate products before peptone is formed in my work on "Diseases of the Stomach" (second edition, pp. 48 and 49; see also J. Reynolds Green, "The Soluble Ferments and Fermentation").

---

* "Die Reactionen des ungeformten Ferments," "Zeitschr. f. physiol. Chem.," 1892, S. 271.

Claude Bernard held the view that the action of pepsin was a preliminary stage in the proteolytic digestion,—a preparation for the more vigorous transformation brought about by the action of trypsin in the intestines—and recent investigations of Pawlow (*l. c.*) and Rachford (*l. c.*) apparently demonstrate an intimate correlation of the two kinds of proteolysis and a dependence of the pancreatic upon the gastric proteolysis. Chittenden and Amerman believe that complete peptonization is not a property of gastric digestion in either the natural or artificial process. The relative amount of proteoses and peptone found when digestion by pepsin and HCl approaches its completion is approximately 65 of proteoses and 30 of peptone—that is, there is more than twice as much of the primary proteoses formed than of subsequent products.

The principal reason why the subsequent action is so slow and incomplete is to be sought in the very presence of the excess of primary products of pepsin and HCl digestion. If the subsequent formation of products of pepsin (heteroproteose, deuteroproteose, amphopeptone) is inhibited in the stomach by the primary products of digestion, it must, of necessity, be much more inhibited in a glass vessel during artificial digestion experiments under conditions as those in which Fleischer's experiments were conducted; for in the stomach the excess of primary products is removed by absorption, and a relation is maintained in which the ferment is enabled to continue its work at least for a longer time than in a glass vessel, where there is no absorption and the accumulation of primary proteoses, together with such other subsequent products as may be formed, must eventually arrest the action of pepsin and HCl entirely.

In determining critically the value of the experimental facts advanced by Fleischer concerning the digestibility and utilization of such proteid foods as calf's-brain, sweetbreads, ham, etc., I fail to recognize that scientific conclusions can be drawn from them, because a very active proteolysis (such as takes place by action of pepsin and HCl on sweetbreads) is very soon arrested by formation of the primary proteoses, as previously described, and the finding of undigested ham and sweetbreads in the digestive mixtures at the end of the experiment does not necessarily prove that these foods are indigestible—it may, indeed, be interpreted as an evidence that they are very readily digested; so readily indeed, that the primary proteoses and subsequent products are so rapidly formed as to arrest the proteolysis at an early stage.

If such mixtures in which the pepsin proteolysis is arrested are placed in dialyzers and kept at proper temperature, so that the soluble products can largely be removed, the digestive process will gradually be resumed and carried to a stage further advanced than was the case in the glass vessel.

Penzoldt's first attempt at establishing a conception of digestibility was by determining the time in which foods were evacuated from the stomach into the intestines. Fleischer's experiments aimed at the same problem by artificial digestion experiments. Both plans, although they have thrown valuable light on this subject, are technically defective, and conclusions of scientific value in explanation of human gastro-intestinal digestion are as yet not matured.

I have already expressed the opinion that the imperfect albumin digestion (proteolysis) in gastric superacidity is due most likely to similar conditions, as are explained in the preceding—that is, the excess of HCl (0.6 per mille and more) causes such rapid formation of primary proteoses (acid albumin) and subsequent products that proteolysis becomes arrested (Schwann, "Künstlich. Verdauung d. geronenen Eiweises," "Müller's Archiv," 1836, 66; also, "Annal. d. Scienc. Nat. et Zool.," 1837, p. 313).

Importance of Spices and Relishes in Nutrition and Digestion.

The spices which are usually added to the food—as, for instance, pepper, mustard, cloves, nutmegs, anise, cumin (or caraway), cinnamon, vanilla, ginger, and onions, the numerous pot-herbs, etc.—are not food-stuffs, but simply relishes. Still they play an important rôle in the nourishment of both the healthy and the sick, because they not only give to the articles of diet the necessary agreeable taste, but, on the other hand, they affect the salivary nerves, and probably also in a reflex way the sensory, motor, vasomotor, and secretory nerves of the digestive organs. The salivary glands are influenced more strongly, and thus the spices materially aid the transformation and assimilation of food-stuffs in the stomach and intestines. Foods that do not have an agreeable taste cause repulsion even in persons who are very hungry, and also cause vomiting and diarrhea, even if they are easily digested. Voit was the first one who rightly appreciated the great importance of the spices and showed their indispensability for nourishment, a fact before contested. The different tastes of men in different countries, as well as within the same nation, may be largely accounted for by the great number of different condiments and the degree to which they are employed. Salt, sugar, and certain organic acids—acetic acid (vinegar), citric acid, lactic acid, and others—are usually classed among the condiments, which also serve as nourishment directly, since they are consumed in large quantities, especially sugar, and thus are at the same time foods and relishes (Voit). The great value of common salt has been known to man for a long time. Indeed, salt has repeatedly served as money among primitive people, and even now it is an important article of exchange. From certain experiments on animals it seems to be proved that salt, if more of it is used than is absolutely necessary, has a favorable effect on the body (Voit). If a diet is very deficient in salt, the body retains the salt which is already present in the blood and in the tissues for a very long time, a proof of its great importance for the organism. On certain forms of bacteria and fungi salt acts like a mild antiseptic. While it does not destroy these organisms absolutely, it at least arrests their growth. Hence salted food is not so decomposable as that which is not salted.

On the basis of the preceding arguments the following demands are to be made of a proper, easily digested nourishment: It must contain, besides water and certain mineral matters, albumin, fats, and carbohydrates, and further, the amount of nitrogenous substances (albumin and gelatin) should be to the nonnitrogenous (fats and carbohydrates) during work as 1 is to 4; during rest, as 1 is to 3.6. If the food is composed in these proportions and reaches the stomach in the proper form and preparation, a considerable part of the albumin and carbohydrates will be digested and absorbed in the stomach, and thus the work of the intestines will be much lessened. Every excessive favoring of the albuminates, fats, or carbohydrates is to be avoided, unless especially valid reasons make it necessary, because the assimilation of the food might be unfavorably affected in the stomach and intestines thereby. The foods must arrive in the stomach in such a form that the food-stuffs contained therein may be most thoroughly assimilated, and at the same time their transformation and absorption should not make excessive demands on the digestive organs. A rational preparation of the vegetable and animal food-stuffs may contribute very much to their better utilization. The more carefully the solid particles of foods are masticated and insalivated, the greater the surface which they offer to the action of the gastric and intestinal digestive juices, the more rapidly they will disintegrate in the stomach and become reduced to smaller and smaller particles or to a pulp; this condition being essential for the transformation and absorption of the nourishing substances contained in them, and consequently for their utilization. Experience and practice are required in the kitchen in the preparation of most vegetable foods. The numerous enveloping membranes, the shells, the bran, and the cuticle in seeds especially, which consist largely of cellulose, surrounding the albuminates and carbohydrates of the vegetable foods and rendering the penetration of the digestive juices difficult or impossible, must be loosened, burst, if possible removed entirely, as by straining through fine sieves. The presence of a certain amount of cellulose in vegetable foods, provided it does not inclose the food-stuffs on all sides, is generally rather useful than harmful, because it stimulates mechanically the mucous membrane of the stomach, and especially that of the intestines, and produces energetic contraction of the muscles, by which the transformation and absorption of food-stuffs are advanced, and the evacuation of the feces promoted. The organic acids introduced with the foods and also those formed in the intestines from the carbohydrates, if they are not present to excess, have the same favorable effect. The method of preparing vegetable foods has a great influence on the utilization of the food substances contained in them. This is proved by the experiments of Rubner, Praussnitz, Weiske, Boeck, and others. Thus, for instance, of the albumin of soaked peas, only 72 per cent.; of that of whole potatoes, but 68 per cent.; while of the albumin of mashed peas, 82 per cent. (that is, 10 per cent. more), of mashed potatoes, 80 per cent. (that is, 12 per cent. more)—are absorbed in the stomach and intestines.

Concerning the utilization of different kinds of bread, it might be emphasized that wheaten bread gives the highest caloric values, as

78 per cent. of the proteids and 99 per cent. of the carbohydrates contained in it are assimilated (Hemmeter, "The Dietetics of Bread and Butter," "Maryland Med. Journal," February, 1900), while rye bread, as well as pumpernickel, are less digestible—for the former, 68 per cent. of the albumin, 89 per cent. of the carbohydrates are assimilated. Macaroni (albumin, 83 per cent., carbohydrates, 99 per cent.), dumplings made of any carbohydrate, noodles, and also foods composed of rice and maize, rice gruel, rice cakes, and corn cakes, are well assimilated and utilized, and are therefore especially suited for the nourishment of convalescents. Corn cakes should be made only on a soapstone griddle and little lard used. Those foods which are composed of the flour of leguminous plants (peas, beans, lentils) having a high nutritive value on account of their great percentages of proteid, are very well adapted to the nourishment of invalids, but only when they are prepared in the proper way. The following interesting observations were made by Strümpell on his own person. In one experiment he ate the flour of legumes in the form of cakes, which were prepared with weighed amounts of milk, butter, and eggs; in a second experiment he consumed lentils which had only been soaked in water and then boiled. Of the albumin of the leguminous substances in the form of cakes, 91.8 per cent. was assimilated; of the cooked unground lentils, only 59.8 per cent. There are numerous examples of irrational and improper food preparations in the daily diets of the lower classes—for instance, a diet consisting principally of potatoes, consumed in great quantities in the form of half-cooked pieces. Since the amount of gastric juice and saliva is insufficient for the great amount of the food to be digested, particles of potatoes can not be penetrated thoroughly and do not disintegrate in the stomach. The larger pieces of potato are evacuated into the intestine only slightly altered, and almost the entire work of digestion devolves upon it. In the jejunum organic acids are formed from the starch of the potato, and the transformation and absorption of the nutritive constituents of the same is still further lessened by the fact that these organic acids, when formed in excessive quantities, may in some patients cause a great increase in the intestinal peristalsis. The sojourn of the chyme in the intestines is too short; both the starch and albumin of the potatoes, as well as other elements of the

food, are passed off undigested in considerable amounts in the profuse evacuations, which may occur several times during the day, and in which undigested particles are often found. A portion of the potatoes should always be consumed in the form of soup or mashed. If circumstances are such that the largest part of the sustenance must be derived therefrom, in the case of sensitive patients they are to be well grated and finally boiled with milk or broth or made into dumplings, cakes, and only a part made use of in the form of thoroughly boiled potatoes, which must be carefully chewed and insalivated in the mouth, so that they may quickly break up in the stomach. Crisped cabbage, and other forms of cabbage, celery, beets, carrots, and turnips are but scantily assimilated in the stomach and intestines; they should not be consumed by dyspeptics; cauliflower, however, is permissible.

As a general rule, the meat of young animals can be eaten soon after they are killed, but that of older animals should first be hung for several weeks. Marine animals should, whenever possible, be brought into the kitchen in a living condition. When oysters are eaten without chewing, the animal continues to live for a while in the consumer's stomach. Crabs, lobsters, and mussels should be put on the stove alive. Fish, whenever possible, should be cooked on the same day they are caught. The meat of older animals (including old hens, pigeons, partridges, hares) should be eaten some time after their death; at any rate not until after rigor has passed away. By means of hard beating, chopping, or scraping, by putting in vinegar, sour milk, or sauer-kraut (lactic acid), the digestibility of the tough flesh, rich in connective tissue, may often be much improved. According to Fleischer, meat that has been smoked too much and too long (especially ham) is not assimilated as well as slightly smoked meat; long-continued roasting of meats is also not advisable. Von Leube recommends roast beef in a rare state, sirloin steak beaten, scraped, or done rare (English style), and raw ham scraped, so that the structure of the connective tissue is removed, as easily digested foods for the nourishment of invalids.

According to Moritz ("Grundzüge d. Kranken-Ernährung," S. 125), cheese is not only a food, but also a digestive aid, stimulating gastric secretion in a reflex way; he thus approves of the custom of closing large meals by eating small pieces of cheese. Since it is a matter of experience, confirmed by analytic experiments, that cheese improves the utilization of milk and of corn meal, and since the same may be true with regard to meat foods, it appears advisable to eat a piece of cheese after a heavy meal. The following utilization of milk alone was determined in a dietetic experiment in which no cheese was eaten: 90 per cent. of the albumin, 94 per cent. of the fats, and 100 per cent. of the carbohydrates are assimilated; after the addition of some cheese, 96 per cent. of the albumin and 97 per cent. of the fats are absorbed; when cheese was eaten with eggs, 97 per cent. of the albumin and 97 per cent. of the fats were taken up. Whether cheese is a dietetic antiseptic and useful to combat the effects of intestinal autointoxication, as A. Lockhart Gillespie suggests (" Remarks on Dyspepsia and Diet," " Brit. Med. Assoc.," July, 1898), is in my opinion doubtful. Gillespie recommends cheese for these conditions because a large number of vegetable and animal micro-organisms are thereby introduced into the intestinal canal, which inhibit putrefaction by the production of organic acids. The intestinal tract requires little aid of a bacterial character—it is already overcrowded in conditions of autointoxication; at least I have not observed that the indican was reduced in amount, nor the ratio of the preformed to the combined sulphates lowered by a cheese diet, these being our chief indications of what has been designated as "intestinal autointoxication."

Those fats are most to be recommended which are liquid at the temperature of the body, because, according to reliable experiments, 98 per cent. to 99 per cent. of these are absorbed. Ten per cent. of those fats which are semisolid at the temperature of the body (beef tallow) pass off unutilized; therefore they are to be consumed only in slight amounts. Those fats which at the temperature of the body are solid are to be entirely avoided, because only from 11 to 13 per cent. of them are absorbed, according to Voit.

Results of Hypernutrition.

The amount of food consumed at the various meals will vary with the climate, seasons, age of the individual, and the work required of him, but it should not be excessively large. This important rule of living is very often ignored, and it has repeatedly been emphasized in my work on "Diseases of the Stomach" that Americans, as a rule, eat much more than is requisite for the appeasing of their hunger, for covering the expenditures of the body, and for the collection of a sufficient amount of reserve fat. If the amount of food digested is so large that it fills the digestive organs to excess, these organs must work excessively for a longer period of time, and their walls are very much distended. If so great an overloading of the stomach and intestines occurs very often, it may cause functional disorders, affecting first the motility, especially if but a short period of rest is granted after hard work, not to mention the more serious consequences—as, for instance, gastritis, enteritis, dilation of the stomach, etc. If the blood becomes overcharged with food substances, great amounts of oxygen are required for its combustion, and also considerable amounts of water in order to separate the excremental matter, especially the urea, through the kidneys. Excessive demands are made on the lungs, the heart, the involuntary muscles of the blood-vessels, the liver, and the kidneys, and it can easily be understood why a long-continued excessive nourishment can cause not only diseased conditions of the digestive organs, but also of the heart (hypertrophy), the blood-vessels (arterial sclerosis), the liver, the kidneys, and even diabetes mellitus and gout. A diet slightly short of caloric requirements is not so dangerous as excessive nourishment, especially if the consumer performs but little mental or physical work, while hypernutrition, on the contrary, is perfectly justified in persons convalescing from long-continued fevers or other sicknesses, if they have lost considerable bodily weight, as well as in the case of persons who have experienced exhaustive hemorrhages. In the latter cases, however, a long period of rest ought to be given to the stomach and intestines after each heavy meal. Palatability and variety are indispensable requisites to a rational diet. Foods which at first were consumed with pleasure, often cause repulsion if repeated too frequently—the more characteristic their flavor, the sooner will the consumer tire of the diet. This is especially true of insipid foods. Palatability can be largely effected by condiments and spices, but these should not be used when the appetite

is already excessive, nor when there are indicati
hyperacidity, enteritis, or ulcers. In anorexia and atony t
do no harm and may induce the patient to eat food that
otherwise be refused.

CHAPTER VI.

DISTURBANCES IN THE SECRETORY FUNCTIONS OF THE INTESTINES, THE PANCREAS, AND THE LIVER.

Disturbances in the Secretion of the Intestinal Juice (Succus Entericus).

Acute and chronic catarrhal, tuberculous, typhoid, dysenteric, syphilitic, carcinomatous, and toxic inflammations of the mucosa of the large and small intestines probably cause a diminution in the secretion of the intestinal juice, especially if they lead to the formation of extensive ulcers, because inflammatory swelling of the mucous membrane closes the narrow outlets of Lieberkühn's glands. Advanced amyloid degeneration, and also atrophy of the mucous membrane, may permanently diminish the secretion of the intestinal juice or destroy it entirely. It is probable, however, that even in the case of peristaltic insufficiency (atony) less intestinal juice is secreted than during normal motility, because in the former case the chyme in its separate parts is not brought into such intimate contact with the intestinal mucosa, and consequently the glands of Lieberkühn are less stimulated to activity by the mechanical and chemical irritants.

Asiatic cholera causes an excessive secretion of the intestinal juice.

Detrimental Results of Decrease in the Secretion of the Intestinal Juice for the Digestion and Absorption of Foods.

Those intestinal affections which lessen the secretion of the intestinal juice usually also cause disturbances in the peristalsis and absorption, and often inhibit or arrest entirely the flow of the bile and of the pancreatic juice into the intestines, so that usually it is impossible to ascertain which of the disturbances in digestion and in absorption, and also which of the changes in the composition of the feces, to ascribe to the lessened secretion of the intestinal juice.

The secretion of the glands of Lieberkühn contains about 0.5 per cent. of sodium chlorid and 0.5 per cent. sodium carbonate. Bunge regards the latter as the most important constituent, because the acidity of the gastric chyme may be readily neutralized thereby and the proper medium brought about for the action of the ferments of the pancreatic juice.

If this view of the neutralizing effect of the intestinal juice is correct, a reduction or arrest of its secretion will disturb the emulsion of the fats, and afterward the transformation and absorption of the other food-stuffs, because the partial or complete lack of the alkali of the intestinal juice hinders the passing over of the chyme into the mildly acid or neutral reaction, which reaction is especially favorable for pancreatic digestion.

Disturbances in the Formation of Bile and in its Flow into the Intestines.

Disturbances in the formation of bile may be caused by a number of hepatic diseases, such as inflammations, neoplasms, fatty or amyloid degeneration, and atrophy. The flow of bile into the intestines may be disturbed by a partial or complete displacement of the larger bile-passages and especially of the common gall-duct. The most frequent cause of stoppage in the ductus choledochus is duodenitis or gastroduodenal catarrh. If the inflammation of the duodenal mucosa extends to the diverticulum of Vater, which is the terminal expansion of the ductus choledochus, then a moderate inflammatory swelling of its mucous membrane, together with a gathering of mucus, may stenose the duct and hinder the outflow of the bile into the intestines. A very slight increase in resistance may cause complete cessation of the flow of bile, because the pressure under which it is contained in the bile-passages is very low. The secretion of the bile into the intestines may be diminished or rendered entirely impossible by an extended catarrh of the larger and smaller bile-passages (cholangitis), as is so frequently seen in connection with cholelithiasis and certain poisonings, also by gall-stones, which become impacted in the bile-passages and especially in the common gall-duct. The latter may be compressed by enlarged lymphatic glands (at the porta of the liver) and by swellings of neighboring organs (especially of the intestines, the pancreas, the gall-bladder, and the kidney).

If the obstruction is situated in the upper part of the common gall-duct, the flow of bile alone into the intestines is hindered; if, however, it is near its opening into the intestine, the pancreatic duct, which is very close to the bile-passage (at the duodenal papilla), or which may even empty into it, is blocked and the secretion of the pancreatic juice into the intestine ceases if there is no second outlet passage for the pancreas (an anatomical variation occasionally observed—ductus Santorini) or if this is also closed (Hemmeter, "Diagnosis and Treatment of Icterus and Cholangitis," "Bulletin of the Hospital of the University of Maryland," March, 1898, p. 27).

Injurious Results of an Obstructed Flow of the Bile into the Intestines, for the Digestion and Absorption of Foods.

In another chapter I have described the work which the bile has to perform during digestion; therefore we can now easily deduce the results of its absence. To the bile is ascribed the power of reflexly exciting the muscles of the small intestine to strong contractions, by which the chyme is quickly forced downward; then the progress of the chyme in the small intestine must be delayed, if the bile ceases to be discharged into the intestines, and may undergo putrefaction already in the small intestine, or the putrefaction in the large intestine may reach a much greater intensity. As fats diminish the secretion of gastric juice (Pawlow, *l. c.*), an abundance of fat in the diet will increase intestinal putrefaction, owing to the fact that more of the albumin is decomposed by putrefactive bacteria than normally. This is partly a consequence of its envelopment in undigested fat, which, while it suppresses gastric secretion, prevents the albumin or proteid of the food from being transformed and absorbed. The prevention of proteid digestion by undigested fat is therefore both a mechanical and a chemical interference. In jaundice due to obstruction the putrefactive products which have thus been formed in greater quantities cause a greater irritation of the intestinal mucous membrane, severe diarrhea sets in, which fails to appear if a diet is prescribed that is free from fats (Stadelmann), and in this case the intestinal putrefaction soon decreases.

When bile is prevented from entering the intestines, the feces are usually very hard, give off a penetrating, decaying odor, especially if the diet has been rich in fats; the air breathed out by patients suffering with jaundice due to obstruction is also very offensive; but this, in my opinion, is largely due to accompanying stomatitis. Instead of the characteristic dark-brown color, the feces have a grayish-white or white appearance, like clay. Hitherto this has generally been explained by the absence of the bile coloring-matter, but, according to Bunge, it is a result of the great amount of fats contained in the stool. According to his experiments, the dark-brown color of the feces, which should be conditioned by sulphid of iron, returns after the extraction of the feces by means of ether, as well as after the introduction of a diet free from fat.

The absence of the weak diastatic effects of the bile probably has no bad effect upon the transformation of the starch in the intestines. If, however, the flow of the bile into the intestines ceases entirely, the digestion and absorption of the fats are very much disturbed. This is easily understood in the light of the universally recognized importance of the bile for adipolysis. The experiments of Voit, Röhmann, and also Fr. Müller are in accordance with this. They prove that in the case of persons suffering from jaundice due to obstruction, and dogs with biliary fistula, about one-half of the fats taken into the body with the food passes out again with the feces not utilized.

According to the experiments of Voit and Röhmann, dogs under normal conditions absorb nearly 99 per cent. of the 150 to 200 gm. of fat fed to them, while dogs suffering from biliary fistula utilize only from 40 to 50 per cent. of the fats in the food. The more fat is introduced in the food, the greater becomes the amount of fat contained in the feces. We must here consider well the fact that in the case of dogs suffering from biliary fistula the larger part of the undigested fat in the feces consists of the fatty acids, not of neutral fats, for this proves that a considerable amount of the neutral fats were decomposed both by the pancreatic juice and also by putrefaction. At any rate, if the flow of the bile into the intestines is hindered, the emulsion and absorption of fats are considerably reduced. In those frequent cases where jaundice due to obstruction occurs in connection with acute enteritis (intestinal catarrh), the disturbance in the emulsification of the fats can partly be traced to the fact that the primary disease caused a diminution in the secretion of the intestinal juice, which is essential to the digestion and absorption by the amount of alkali contained in it. The consumption of fatty foods must, therefore, be strictly forbidden to those patients suffering from jaundice due to obstruction not only because only a small part of the same is absorbed in the intestines, but also because it increases the intestinal putrefaction, causes a disagreeable flatulency, and lessens the digestion and absorption of the albumin or proteids of the food.

The investigations in cases of chronic fatty diarrhea of children, carried out by Baginsky, Biedert, Demme, and others, prove that nutrition may be very much affected by the disturbances in the transformation and absorption of fats. The little patients soon become emaciated, and finally succumb to marasmus. In one pronounced case of fatty diarrhea Biedert found in the autopsy the signs of gastroduodenal catarrh, and more especially a large inflammatory swelling in the neighborhood of the outlet of the common gall-duct and of the pancreatic duct into the intestines, which had doubtless hindered the outflow of the bile and of the pancreatic juice during life.

Disturbances in the Secretion of the Pancreatic Juice.

While disturbances in the formation and outflow of the bile are observed quite often, it is very seldom that cases are observed where the secretion of the pancreatic juice is diminished or arrested, or where its outflow into the intestines is hindered or completely checked without any simultaneous disturbance in the outflow of the bile. The secretion of the pancreatic juice may be disturbed and even stopped by a partial or total degeneration, by interstitial inflammation with consequent atrophy, which latter is also occasionally caused by the formation of concretions—pancreatic calculi—which exert a strong pressure on the secreting tissues, and also by tumors, neoplasms, and cysts of the pancreas. The outflow of the pancreatic juice may be hindered or entirely stopped by secondary inflammations of the collecting and discharging passages, subsequent to an intestinal catarrh; more rarely by concretions which have become lodged in these passages.

The most frequent cause of a diminution in the amount of pancreatic juice in the intestines is the closing of the outlet of the pancreatic duct (ductus Wirsungianus) at the duodenal papilla. In such cases, however, the bile is also always lacking in the intestines. If the gland has but a single outlet passage,—that is, the duct of Wirsung,—or if the second outlet, the duct of Santorini, is also closed as a result of catarrh, the outflow of the pancreatic juice into the intestines naturally ceases entirely.

Results of the Absence of the Pancreatic Juice for the Digestion of Food Substances.

Interference with or arrest of the flow of pancreatic juice without simultaneous interference with the secretion of the bile is rarely observed. This is due largely to the anatomical fact that the orifices of both ducts are located close to each other on the duodenal papilla, and a disease attacking one is likely to involve the other. At one time it was held by noted physiologists that adipolysis was carried on by the pancreatic juice alone, and also that all fats and fatty acids which were liquid at the temperature of the body were emulsified by it. From this it was a natural deduction that an abnormally high proportion of fats in the feces had to be considered a valuable symptom of a disease of the pancreas or of a closing of the outlet passage of the gland. Since it has been ascertained, however, that the bile also may decompose neutral fats, and that the pancreatic juice is not concerned actively in their emulsification in a nourishment consisting of albumin and fats, but only indirectly, and since it is known that this emulsification is caused principally by the intestinal juice, assisted by the bile, this symptom, so far as it has reference to the pancreas, has lost its former importance. The experiments of Fr. Müller confirm this view and show that, in the case of patients suffering from advanced degeneration of the gland, the digestion and absorption of the fats were but little disturbed. The experiments of Müller could be carried on for but a short time, and it is to be regretted that longer experiments on his patients in food utilization after the introduction of abundant amounts of albumin and fats could not be carried out. On this account they lose their cogency, especially since there is a possibility that some pancreatic juice, even if in diminished quantity, was nevertheless secreted into the intestine in spite of the extensive disease of the pancreas. This source of error can never be entirely excluded in the experiments on persons suffering from diseases of the pancreas entirely occluding the pancreatic duct. By the author's method of duodenal intubation it was possible in one case to establish absence of pancreatic secretion from the duodenum. This conclusion was based on the fact observed, that digestive tests with filtrates of the drawn duodenal contents gave no evidence of proteolytic ferment that was active in alkaline solution after filtration of the duodenal chyme through a Pasteur filter to exclude the possible proteolytic action of bacteria. If the mouth of the pancreatic duct is blocked, pancreatic juice may still flow into the intestines through the duct of Santorini. If this latter outlet is wanting or is also closed, the flow of the pancreatic juice into the intestine ceases entirely, but we can not prove it, except in rare and fortunate cases, by duodenal intubation ("Intubation des Duodenum," Hemmeter, "Arch. f. Verdauungs-Krankheiten," Bd. 11, S. 85). Experiments have been made to extirpate the pancreas entirely in animals, in order to keep the pancreatic juice out of the intestines altogether, and thus ascertain definitely the bad results of its absence upon the digestion of food-stuffs. Utilization experiments on animals the pancreas of which had been removed gave the following results: The neutral fats introduced in the foods are decomposed (from 30 to 85 per cent.), but appear again in the feces, and only about 44 per cent. of the albuminates and from 60 to 80 per cent. of the carbohydrates were assimilated in the intestines (Abelmann). Extirpation of the pancreas is a very severe abdominal operation, and part of the disastrous results following it may be ascribable to the "*pancreatic icterus*" referred to in the chapter on Intestinal Dystrypsia. There is good reason for believing that by it the amount and composition of the bile might be affected. In the second place, after this extirpation, severe melituria sets in and continues for a long time, which may not only affect the absorption and peristalsis of the intestines, but also the secretion of the bile and of the intestinal juice. Schiff's experiments proved that animals whose pancreatic duct had been ligated, and after which melituria did not set in (see Minkowski, Mering, and others), still digested and absorbed from 150 to 200 gm. fats. The utilization experiments after the removal of the pancreas ought to be repeated with this modification—namely, a very small portion of the pancreas should be preserved, for then melituria does not set in, although the secretion of the pancreatic juice ceases. Following upon ligation of the pancreatic duct we might also expect a greater disturbance in the digestion and absorption of fats, because then the HCl of the gastric juice, which otherwise is neutralized by the alkalis of the pancreatic juice, unites with a portion of the alkali of the intestinal juice and thus weakens the emulsifying power of the latter. The statement that animals whose pancreas had been entirely removed and in whom all neutral fats appeared again in the feces, nevertheless could absorb from 30 to 50 per cent. of these fats if they were taken in as a natural emulsion (milk), argues for an injurious effect on the emulsification and saponification of fats when the pancreatic juice is entirely lacking. Possibly in the case of stenosis of the pancreatic duct a readjustment is made in the chemical reaction of the total intestinal secretions by an increase in the alkalinity of the intestinal juice, perhaps also of the bile; besides, a portion of the pancreatic juice may be secreted into the intestine through the duct of Santorini, even after the pancreatic duct has been ligated or stenosed.

It has been emphasized previously that if the mouth of the pancreatic duct at the duodenal papilla has been stenosed, the bile is also always lacking in the intestines. In this case the digestion and absorption of the fats must be very much impaired, especially if the pancreas has but one outlet or if the duct of Santorini is also closed. Since only a small part of the fats is decomposed by putrefaction when the outlet passages of the pancreas and of the common gall-duct are closed at the same time, we may expect that in such cases most of the neutral fats would appear again as such—that is, undecomposed—in the feces, whereas if the common gall-duct alone is blocked, the fats should be carried off in the feces as fatty acids. If this assumption be confirmed in the future by means of exact experiments, we should have gained a certain fixed basis for the recognition of the simultaneous closure of both the outlet passages so frequently referred to in this section.

It may be possible to discover whether the feces contain large amounts of fatty acids, when both ducts are stenosed. In that case the assumption seems justified that those fat-decomposing ferments retained in the bile-passages and the outlet passages of the pancreas are again secreted with the intestinal juice after their return into the blood, just as was supposed to be the case with pancreatic ptyalin previously referred to. If this supposition proves to be correct, it would be a demonstration of the manifold ways in which the various ferments of the digestive glands of the intestines may mutually replace each other—for instance, if the amylopsin or the alkali of the pancreatic juice is not permitted to be active because, for some reason, the outflow of this secretion is prevented, then there will be more diastase and alkali in the secretion of the glands of Lieberkühn, but to my mind this admirable readjustment and equalization arrangement is not as yet satisfactorily proved.

As to the transformation of the *albuminates* and *carbohydrates* (starch), when the secretion of the pancreatic juice is lessened or stopped the experiments of Fr. Müller prove that in the case of patients suffering from advanced degeneration of the pancreas the transformation and absorption of albuminates are disturbed very little. The conversion of the carbohydrates is not at all affected. In the case of animals, however, whose pancreas has been removed, only 44 per cent. of the albuminates and from 60 to 80 per cent. of the carbohydrates were absorbed. The impairment of the tryptic albumin digestion is very probably compensated for the most part by the fact that more albumin than usual is changed in the duodenum into syntonin and peptone by the pepsin and HCl of the gastric juice, because the neutralization of the HCl in the intestine occurs later than during normal digestion, on account of the partial lack of the alkali. Another explanation is that referred to in the preceding—namely, that the succus entericus in those cases of loss or destruction of the pancreas will contain more proteolytic ferment.

It is more difficult to explain the statement that the transformation of the carbohydrates is not detrimentally influenced when the pancreatic juice is much decreased, for its diastatic efficacy is known to be very strong. The supposition has already been advanced that when the pancreatic duct is closed up, the pancreatic ptyalin retained in the gland may possibly be secreted into the intestine with the intestinal juice after its entrance into the blood (finally also with the bile), and thus increase diastatic efficacy of these secretions. This assumption is supported by the fact that in those animals whose pancreas has been removed and in whom no pancreatic ptyalin can be at all formed, only from 60 to 80 per cent. of the amylaceous substances introduced are absorbed. As long, however, as we are not certain whether the melituria occurring after the removal of the pancreas affects the transformation favorably or not, the result of these animal experiments is ambiguous, and therefore can not be considered as a reliable proof. Other utilization experiments must, therefore, be made on animals whose pancreas has not been entirely removed, and in whom melituria has thus been prevented. By this means it would be possible to determine whether in this case the transformation of the starch in the intestines is impaired to the same degree as when the pancreas is totally removed and melituria ensues.

Since there is no doubt that under normal conditions the pancreatic juice plays an important rôle in the transformation of albuminates, fats, and carbohydrates, and thus also in their absorption in the intestines, further exact experiments on patients suffering from pancreatic abnormalities and on animals are very desirable, so that we may determine to what extent the transformation of these food substances and their absorption in the intestines are affected when the amount of the pancreatic juice is considerably diminished.

The science of the functions of the intestines, as well as of other organs taking part in digestion, and their abnormal variations, form by far the largest and most important part of the pathology of intestinal digestion ; indeed, the former would completely include the latter if bacterial putrefaction, which can not be classed as a function of the intestine, did not have any share in the transformation of the food-stuffs, and if, in the second place, in addition to water and the inorganic salts, only albumin, fats, and carbohydrates in pure condition were introduced, which would be absorbed in the intestine without residue. This is, however, not the case ; a portion of the three food-stuffs, although a very small portion, is transformed by bacteria, and, besides, most of the food substances, both animal and especially vegetable, contain matter which is very difficult or impossible to digest, and which passes forth from the body unutilized. That plants contain a much larger proportion of indigestible matter is proved by the fact that if the diet consists mainly of vegetables, the evacuations occur several times during the day, and are more voluminous than those resulting from a diet consisting of meat alone, when evacuation occurs usually but once a day. The cause of the incomplete utilization of the vegetable foods is assigned to the greater or lesser amount of cellulose contained in them. Tappeiner and others have proved that the old assumption that cellulose can be digested and absorbed only in the gastro-intestinal canal of the herbivorous animals, where the ingesta remain for a longer period of time, is false. These experiments showed that cellulose may also disappear in the intestines of man, although the opinions vary as to whether this transformation is due to putrefactive bacteria or intestinal ferments. At the same time only a very small portion, probably, is absorbed; the principal part passes off with the feces. Besides this, cellulose also renders the utilization of the vegetable foods more difficult by inclosing the nutritious parts of these foods (albumin and starch) with shells or cuticles, and thus preventing the action of the digestive juices upon them. The result is that a portion of the food is not digested and absorbed and is carried off with the feces. When we discussed the disadvantages of a diet consisting principally of potatoes, it was stated that in the very abundant evacuations, undigested starch and even entirely unchanged particles of potatoes, and also other food particles, may be found in greater or less abundance besides the organic acids which might have been utilized in the economy of the body. If our nutrition, therefore, is to be rational, it is not enough to introduce a sufficient amount of food: it must be offered to the digestive organs in such a form that it may be transformed and absorbed as thoroughly as possible without too great exertion of the glandular and muscular apparatus.

The manner in which the various food and dietetic substances may be combined so as to aid each other mutually in their digestion, and the way in which food substances act as physiological stimuli to specific gland cells has been admirably set forth in the work of Pawlow ("Die Arbeit der Verdauungsdrüsen," S. 173). This accurate physiologist has emphasized that the beginning of the secretory activity of the stomach is dependent on the psychic condition which harmonizes with the demands of every-day life, that the food should be eaten with attention and pleasure. I shall consider the effects of various foods as specific dietetic stimuli to definite digestive gland-cells in another chapter.

CHAPTER VII.

THE INTESTINAL BACTERIA.

BY WM. ROYAL STOKES, M.D.

In describing the bacteria of the intestines, it is not sufficient merely to enumerate the species which are usually present. It is also important to describe the organisms found in intestinal diseases and their relation to the lesions produced, as well as to consider the part played by various bacteria in the processes of digestion.

First Observations.

Bacteria were first observed in the intestinal contents by Van Leuwenhoek in 1675, but he incorrectly considered them as belonging to the animal kingdom. He named them "animalculæ," and was influenced in this opinion by the activity which many of these organisms displayed when viewed under the lens of his primitive microscope.

Billroth,[1] nearly two centuries later, observed many germs in the intestinal contents, which he described under the general name of coccobacteria. Nothnagel[2] soon followed with the opinion that these intestinal bacteria were concerned in the decomposition and fermentation of albuminous bodies in the intestine, and he mentioned such organisms as Bacillus subtilis, streptococci, saccharomyces, and Bacillus butyricus as normal inhabitants of the intestine. Before considering the more modern work on the subject, it will be necessary to mention some general facts concerning the bacteria of the intestines.

Fetal Feces.

It has been shown by Escherich[31] that the meconium of the fetus is sterile, but the intestinal contents become infected by bacteria even before the infant takes any nourishment from the mother. The swallowing of air and infection from the maternal parts during birth probably cause the introduction of bacteria. These soon increase in the intestinal contents, and Escherich has cultivated many cocci, bacilli, and yeast cells from the feces only twenty-four hours after birth.

Numbers.

The numbers of bacteria in the intestine vary greatly, and seem to be influenced by the taking of food and the voiding of feces. Von Sucksdorff[3] counted the intestinal bacteria in a milligram daily for twenty-four days, and found a maximum number of 2,300,000, a minimum number of 25,000, with a general average of 381,000 bacteria in a milligram of fecal matter. Figuring upon the entire quantity of feces passed in twenty-four hours, he came to the conclusion that an average of 53,124,000 bacteria were passed daily by a healthy person. The number of bacteria can be decreased by the use of sterilized food to 10,395 bacteria in a milligram, but the contents of the large intestine can not be rendered sterile by this method. The majority of bacteria are, therefore, introduced by means of the food. The mouth also contains a large number of bacteria, and these are often swallowed with the saliva, and many survive in the intestinal tract. Miller[4] has demonstrated eight varieties of the bacteria of the mouth in the stomach-contents, and twelve varieties of mouth bacteria in the feces, but it is generally held that only about three per cent. of the intestinal bacteria are derived from the mouth, and the other 97 per cent. are obtained from the food.

These facts disprove the old idea that the gastric juice is a strong germicide which quickly destroys bacteria when they enter the stomach. A number of investigators have proved that bacteria can pass unchanged through the stomach, and that this organ is rarely entirely free from bacteria. Staphylococcus pyogenes aureus, Bacillus tuberculosis, and Bacillus diphtheriæ can pass through the stomach without change.

The large intestines contain more bacteria than the small intestines, with the exception of the lowest portion of the ileum.

Studying the Intestinal Flora.

In order to study the intestinal bacteria it is necessary to obtain a small quantity of feces, dilute it in water, and then study these organisms in a fresh condition under the microscope by means of the concave glass slide. The various sizes and shapes of the organisms, as well as the motility, can thus be studied.

A specimen should also be mounted on a cover-slip, dried, and stained by an anilin dye, such as fuchsin or methylene-blue, and by Gram's method, in order to determine the morphology and various staining reactions.

Culture Methods.

The most valuable method, however, for studying intestinal bacteria is to obtain pure cultures of the various bacteria, and this is best accomplished by Booker's[6] method.

The intestinal germs are obtained by introducing a sterilized glass tube into the anus, and inserting a longer and smaller sterilized glass tube into the rectum through the first tube. This prevents the contamination of the specimen by the bacteria around the anus, and collects the bacteria only from the interior of the rectum.

The contents of the rectum are soon forced into the inner tube, which is then withdrawn and emptied into a sterile test-tube. The material can then be properly diluted, and plate cultures can be made in neutral glycerin agar. This is better than gelatin, since the latter will soon become liquefied by some of the intestinal organisms. The special and selective media will be mentioned later. Many of the organisms which appear in cover-slips will not grow on our ordinary culture-media, and some varieties have never been cultivated for this reason. ' There are a number of anaerobic organisms present in the intestine, and anaerobic culture methods should always be employed as well as the ordinary methods.

BACILLUS COLI COMMUNIS (ESCHERICH).

Bacillus coli communis is the normal inhabitant of the intestines, and is present in the feces in enormous numbers.

The Colon Group.

Although Escherich[7] and others have used certain cultural differences in order to subdivide the colon bacillus into several distinct varieties, yet there is a certain series of reactions and characteristics which are enough to distinguish the organisms as a species. The following description, therefore, will apply only to the general group or species known as Bacillus coli communis, or colon bacillus group.

Morphology.—The morphology of the colon bacillus is variable, and it often simply consists of a short ovoid body with a diameter of about 0.5 μ. They are at times united in pairs. They often grow out into longer forms, but the average length is from 2 to 3 μ.

Staining Properties.—This bacillus stains by means of the ordinary anilin dyes, but it loses its stain when Gram's method is used. It often takes up the stain irregularly, certain portions remaining unstained. Lateral flagella can be shown by the special stains.

The motility is inconstant, and even when present, it is more sluggish than that of such bacteria as the typhoid bacillus. There are two forms—the motile, consisting of about two-fifths of the group, and the nonmotile, consisting of three-fifths of the isolated bacilli. This motility is, of course, due to the flagella, which propel it through the liquid.

Cultural Characteristics.—No spore formation has been observed, and its thermal death-point is 62° C. for ten minutes. The organism will grow both under aerobic and less favorably under anaerobic conditions, and its growth always produces an acid reaction of the medium if any sugar be present.

The cultural characteristics are typical, and easily serve to distinguish it from the typhoid bacillus and other pathogenic bacteria. When no sugar is present, the reaction is alkaline.

On agar or gelatin plates the deep colonies under the low power are biconvex, or round, often with a teat-like process at one side, and the superficial colonies present no typical structure. These latter appear to the naked eye as elevated, moist, round bodies which are pearl-colored to transmitted light. The puncture culture in gelatin spreads out on the surface as the elevated moist head of a nail, and the streak of growth along the line of needle puncture forms the shank. The gelatin is not liquefied. On slant agar it forms a thick, moist, grayish growth. On potato the bacillus forms a luxuriant growth, often with a few small gas-bubbles on the surface. Litmus milk is acidulated, and therefore turns a decided pink in twenty-four hours, and the medium later coagulates. Bouillon is clouded.

Indol Reaction.—In Dunham's solution a characteristic reaction can be obtained after twenty-four hours' growth in the incubator. This is called the indol reaction, and is obtained in the following manner:

The solution used in the indol test is made by adding one part of H_2SO_4 to ten parts of a 0.02 per cent. solution of KNO_2 (nitrite of potash) in distilled water. This solution of nitrite of potash is made by adding 0.2 gm. of KNO_2 to 100 c.c. of distilled water. Ten cubic centimeters of this solution are added to forty cubic centimeters of water, making a solution of the strength of 0.02 per cent. of nitrite of potash. Nitrite of soda can also be used in making this test.

Ten parts of this solution are *carefully* added to one part of pure H_2SO_4, and one cubic centimeter of this mixture is mixed with ten cubic centimeters of the forty-eight-hour culture of the suspected colon bacillus in Dunham's solution. The indol test will usually appear in about fifteen minutes, but certain varieties of Bacillus coli communis will not give this test until the nitrite solution has remained in the Dunham's solution for twenty-four hours. If indol be present, the solution turns a delicate pink.

Cultures of the colon bacillus have an unpleasant fecal odor, and the bacillus grows well in urine and bile. It can usually be obtained in large numbers in cultures from the bile.

Gas-production.—One of the most important characteristics of Bacillus coli communis is its property of causing fermentation with the formation of gas in various solutions of sugar. This test alone distinguishes it from the typhoid bacillus, and is useful in separating it from other fermentative bacteria.

Theobald Smith[8] has carefully studied this property of the colon bacillus, and after testing a number of colon bacilli from various sources, he concluded that this organism caused a series of characteristic changes when inoculated into the fermentation tube, containing solutions of grape-, milk-, and cane-sugar in ordinary nutrient bouillon. Beef-tea as usually prepared, however, still contains a small amount of muscle sugar, and Smith eliminates this source of error by preparing the nutrient bouillon, and then inoculating it with a culture of the colon bacillus after placing it in a sterile flask.

This mixture is allowed to remain in the thermostat for twenty-four hours, and all the inosite, or muscle sugar, is reduced to other compounds by fermentation. This sugar-free bouillon is then sterilized by steam, or the autoclave, and is then ready for use in the fermentation tube after proper sterilization and filtration.

The colon bacillus will ferment glucose, lactose, and saccharose bouillon in the fermentation tube in a characteristic manner when the bouillon contains 1 per cent. of each of these sugars.

In 1 per cent. glucose bouillon in the fermentation tube this organism will cause the formation of about 50 per cent. of gas in the closed stem in from three to five days. This gas will consist of two parts of carbon dioxid to one part of hydrogen, and the reaction of the medium will always be acid. The proportions of gas can be determined by mixing a 5 per cent. solution of caustic potash or soda with the liquid in the tube, and allowing the thumb to remain on the opening of the tube. On removing the thumb the liquid will rise one-third of the way in the closed stem, and this shows that the gas consisted of one-third part of CO_2, and that the caustic potash changed this into carbonate of potash. The remaining two-thirds of the gas is probably hydrogen, since it can be brought into the open bulb of the fermentation tube and be exploded with a blue flame by applying a match.

On using a 1 per cent. solution of lactose, or milk-sugar, in the fermentation tube, the result is similar to that previously described. On using a 1 per cent. solution of saccharose in bouillon, about one-half of the varieties of the colon bacillus will not cause any fermentation of the fluid, and the other half will cause the formation of about 30 per cent. of gas in from three to five days. The medium may be either acid or alkaline.

This fermentation formula is of great aid in distinguishing the colon bacillus from other pathogenic germs. It has been shown by Fuller that when the colon bacillus becomes degenerated by long exposure to water or other attenuating conditions, the production of gas in the fermentation tube will vary greatly. The work of Theobald Smith, however, has shown that the fermentation formula is fairly constant when the organism is derived directly from the feces, and will serve to identify it under such conditions. The attenuated water bacteria can also be reclaimed by cultivation for several weeks in the laboratory.

If the fermentation tube is not at hand, the gas-formation can be secured by adding 1 per cent. of glucose to a tube of melted agar, and then carefully sterilizing the tube by moist heat. While the agar is still in a melted state, it is reduced to a temperature of 40° C., and is then inoculated with the colon bacillus. This mixture is then placed in the thermostat, and in twenty-four hours numerous gas-bubbles will have formed in the depths of the solid medium.

Pathogenic Properties.—Bacillus coli communis is pathogenic for mice when injected intraperitoneally with from 0.1 to 1 c.c. of a bouillon culture, and the organisms can be recovered from the blood and peritoneal cavity. An enteritis is usually present, and the spleen is enlarged. Large doses will cause general infection in the guinea-pig or rabbit when injected into the peritoneal cavity or blood, but when introduced subcutaneously, only local abscesses are produced. By causing an artificial obstruction to the flow of urine and then injecting the colon bacillus into the urethra of dogs, a cystitis and a pyelonephritis have been produced, and by an obstruction to the flow of bile inflammation of the bile-ducts and even liver abscesses have been induced.

Infection and inflammation of the intestines can be produced, however, only by first injuring or constricting the lumen of the intestine, and then injecting very virulent cultures of the colon bacillus. All this experimental work goes to show that this organism is not pathogenic when it is present in the normal healthy intestine. Under these conditions it simply carries on its normal function of fermenting the various sugars, such as glucose and lactose.

When the intestine is injured or the organism enters an unusual or abnormal situation of the body, such as the peritoneal cavity, it can set up the various lesions previously mentioned. This is its usual course in the human being when causing disease.

Bacillus coli communis is the first organism of decomposition to enter the tissues after death, especially if any intestinal lesions are present, but it can also enter the various organs during life, when intestinal obstruction or extensive necrosis exists. When obstruction to the flow of bile or urine exists, this organism is capable of setting up inflammations of the biliary and urinary tract, and even abscesses in the liver or kidneys. It is said that at times Bacillus coli communis can become increased in virulence and cause an injury to the epithelium, and then enter the tissues of the intestine and even the other internal organs. This causes enteritis, even with ulceration, especially in infants and the young. When the colon bacillus enters the peritoneal cavity through a ruptured or gangrenous appendix or a perforation from an ulcer, diffuse peritonitis occurs, and not infrequently this organism is found in pure culture from the pus, although often other organisms, such as the streptococcus, are present (see chapter on Appendicitis).

Agglutinative Reaction.—When about one milligram of dead colon bacilli is injected into the abdominal cavity of a guinea-pig and the dose is repeated after several days, the guinea-pig becomes immune to infection, and the blood of the pig will cause an agglutination of this colon bacillus in dilutions of one to fifty or even greater dilutions.

Lesage[9] claims that this agglutinative reaction can be used as a sign for infection by means of the colon bacillus, and in forty out of fifty cases of enteritis in infants he found that the agglutinative reaction took place when the cultures obtained from the infants were used, and even when other colon bacilli were tested, though in the later cases only with lower dilutions. Dunham, in the "Journal of Experimental Medicine" for January, 1901, has shown that if animals are treated with gradually increasing doses of dead cultures of the colon bacillus for a long time, a serum with an agglutinative potency of 1 : 50,000 can be secured. He also found, however, that different races of Bacillus coli communis which are entirely similar in every other respect will not give a reaction to any other serum except their own. This failure to give a mutual serum reaction would seem to divide the group into different varieties, as far as their serum reaction is concerned.

BACILLUS LACTIS AËROGENES (ESCHERICH).

Bacillus lactis aërogenes has been demonstrated by Escherich in the intestines and feces of animals and human beings nourished by milk. This bacillus is present in much larger quantities than the colon bacillus in the upper portion of the small intestine, and in the colon the number decreases, only a comparatively small number of these bacilli being found in the stools.

Resemblance to the Colon Bacillus.—Bacillus lactis aërogenes resembles the colon bacillus in most of its morphological, staining, and biological properties, but it exhibits enough differences to distinguish it as a separate species. In the first place, it possesses a viscid capsule which separates individual bacilli when they are massed together in groups. It is invariably nonmotile. Its growth on potato, agar, and gelatin is more profuse, more viscid, and fleshier than that of the colon bacillus, and the cultures give off a more decided fecal odor. Escherich holds that the great characteristic of this bacillus is its property of fermenting the milk-sugar, or lactose, in milk. It causes firm coagulation of the milk in about sixty hours, and 58 per cent. of the lactose present is turned into lactic acid. It also ferments starch.

Pathogenesis.—The injection of large quantities of fluid cultures into the blood of rabbits and guinea-pigs will cause death, and cultures from potato killed by alcohol will poison rabbits when introduced into the peritoneal cavity. The organism is thus seen to be similar to the colon bacillus in its effect upon animals, but it is not known to play any part in human pathology. It may perform an important physiological function, however, by fermenting the milk-sugar present in the small intestine.

FERMENTATION FORMULA OF THE COLON BACILLUS.

The following tables are from Theobald Smith's article on the fermentation tube, and give an excellent idea of the reaction produced by the colon bacillus and Bacillus lactis aërogenes when inoculated into the various sugars in the fermentation tube:

BACILLUS COLI COMMUNIS IN GLUCOSE BOUILLON.—(*Theobald Smith.*)

BACILLUS COLI COMMUNIS.	GAS ACCUMULATED AT THE END OF				CO_2.	H.
	1 Day.	2 Days.	3 Days.	9 Days.		
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
1. From human feces, . .	46	58	62	57	37.2	62.8
2. From human feces, . .	30	45	54	45 (6th)*	37	63
3. From cattle, ...	44	52	62	58	37.2	62.8
4. From swine, ...	36	42	44	46	33	67
5. From water, ...	28	44	47	44	32	68
6. From water, ...	34	47	54 (5th)*	45	31.5	68.5
7. Bacillus of grouse disease (Klein), ...	43	51	53	51 (6th)*	34	66

(* The figures refer to the number of days after inoculation of the culture fluid.)

BACILLUS COLI IN LACTOSE BOUILLON.—(*Smith.*)

Bacillus Coli Communis.	Gas Accumulated at the End of						Total.	CO_2.	H.
	1 Day.	2 Days.	3 Days.	4 Days.	5 Days.	6 Days.			
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
1. From human feces, . . .	. . .	42	58	62	. . .	. . .	60 (11th)	33.8	66.2
2. From human feces, . . .	60	65	. . .	. . .	. . .	66	60 (7th)	. . .	. . .
3. From cattle,	51	56	60	. . .	. . .	61	56 (7th)	36.7	62.3
4. From swine,	39	44	46	.	51	. . .	50 (8th)	40	60
5. From water,	28	42	45	48	. . .	52	45 (8th)	37	63
6. From water,	33	51	56	.	. .	64	59 (11th)	40.5	59.5
6. From water,*	24	54	44	48	50	. .	48 (8th)	35	65
7. Bacillus of grouse disease,	55	62	64	. .	. . .	. . .	61 (6th)	37	63

(* Bouillon entirely free from muscle glucose. Culture made six months after the first.)

BACILLUS COLI IN SACCHAROSE BOUILLON.—(*Smith.*)

Bacillus Coli Communis.	Gas Accumulated at the End of						Total Gas.	Reaction of Bulb.	CO_2.	H.
	2 Days.	4 Days.	6 Days.	7 Days.	9 Days.	12 Days.				
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.		Per cent.	Per cent.
1, . . .	22.4	30	56	63	. .	41 (11th)	60 (11th)	Acid.	38	62
1, . . .	16	.		26	31	58 (13th)	65 (29th)	Alkaline.	43.5	57 5
2, . . .	21	38	46	. .	53	. .	63 (20th)	Acid.	37	63
3, . . .	. .	. .	. .	. .	Bubble.	. .	.	Alkaline.	. .	. .
4, . . .	18	21	26	27	32	36	46 (18th)	Acid.	38 2	61 8
5, . . .	31	47	50	.	. .	. .	43 (11th)	Acid.	36 5	63 5
6*,	7	13	15	16	20 (8th)	. .	19 (10th)	Alkaline.	23	77
6*, . .	.	.	. .	14	. .	. .	13 (10th)	Alkaline.	20	80
7. Grouse disease,	47	50	. .	. .	. .	. .	50 (6th)	Acid.	35.5	64.5

(* Much of the gas set free by this bacillus must be ascribed to muscle glucose.)

BACILLUS LACTIS AËROGENES IN SUGAR BOUILLON.—(*Smith.*)

Kind of Sugar.	2 Days.	3 Days.	4 Days.	5 Days.	6 Days.	Total.	CO_2.	H.	Remarks.
Glucose, . .	41	54	. .	. .	. .	50 (11th)	35	65	Growth subsided. Acid.
	60*	. .	. .	. .	. .	58 (7th)	. .	. .	Growth subsided. Acid.
Lactose, . .	45	61	63	66	68	62 (11th)	38.5	61.5	Growth subsided. Acid.
	40*	47	50	. .	52	53 (14th)	38.7	61.3	Growth subsided. Acid.
Saccharose,	8	9	11	13	. .	13 (11th)	6	94	Growth abundant. Alkaline.
	8*	12	. .	. .	15	17 (13th)	20	80	Growth abundant. Alkaline.

(* The second trials with the same kind of sugar were made fully six months after the first.)

It is now a well-recognized fact that the group of bacteria known as the colon group comprises a large number of varieties with slightly different characteristics, and that the characteristics which have just been mentioned can merely be considered as typical of this group. The colon bacillus exhibits well-marked differences from the typhoid group of bacteria, but an intermediate group exists which possesses characteristics of both of the other groups which have just been mentioned.

Members of this Group.

This intermediate class of bacilli is known as the hog cholera group, and comprises Bacillus enteritidis of Gärtner, the hog cholera bacillus, Bacillus icteroides, Bacillus typhi murium, and a series of organisms which have caused meat poisoning, such as Bacillus morbificus bovis and Bacillus "Hatton." Gwyn also isolated an organism from a case with the clinical history of typhoid fever in which the blood gave no Widal reaction to typical typhoid bacilli, but which gave a Widal reaction when his bacillus was brought in contact with the diluted blood of the patient. This bacillus was isolated from the blood and was called the paracolon bacillus. It evidently belonged to this intermediate group. Cushing[34] has also found a member of the hog cholera group in the pus from a costochondral osteomyelitis which occurred six months after an attack of typhoid fever. This organism is called Bacillus O.

All the organisms which have been enumerated have about the same biological characters, and their properties can be described as follows:

THE INTERMEDIATE, OR HOG CHOLERA, GROUP.

The bacilli are similar in morphology to the typhoid bacillus, and decolorize by Gram's method. They are actively motile, and possess about fourteen flagella.

On the surface of gelatin and agar these bacilli resemble typhoid colonies, being elevated, moist, and bluish to transmitted light. In bouillon the medium is rapidly clouded, and a surface pellicle is usually formed. From 0.1 to 5 per cent. of acid is formed. They all grow anaerobically as well as aerobically.

No indol, or at least only a trace, is formed in Dunham's solution, and only a slight growth takes place on potato. An important reaction of this intermediate group is its effect on milk. This medium is rapidly rendered alkaline until the casein is dissolved and the fat globules can be seen on the surface. The medium finally becomes translucent.

Fermentation Formula.—The reaction in the fermentation tube with these organisms differs both from that of the colon bacillus and the typhoid bacillus.

When the hog cholera group is introduced into dextrose (glucose) bouillon in the fermentation tube, the total gas-production varies from one-third to one-half of total displacement in the closed branch of the tube.

If the initial reaction is 1.5 per cent. of acidity, the group produces a terminal acidity of from 3.5 to about 4 per cent. The proportion of gases is always that of two parts of hydrogen to one of carbon dioxid. The acidity in the closed branch is about 0.20 per cent. in excess of that in the open bulb, and is based upon the amount of decinormal sodium hydroxid required to neutralize ten cubic centimeters of the medium.

In lactose 1 per cent. sugar-free bouillon no gas is formed by the various members of this group, and the same may be said of 1 per cent. saccharose bouillon. In lactose bouillon the reaction in the bulb varies from 0.90 per cent. alkaline to 1.60 per cent., while the closed branch only shows an alkalinity varying from 0.10 per cent. to 0.20 per cent. In saccharose bouillon the reaction in the bulb is from 2 per cent. to 2.4 per cent. alkaline, while the reaction in the closed branch is only 0.2 per cent. alkaline. It will thus be seen that this group produces an acid reaction in dextrose bouillon, but an alkaline reaction in lactose and saccharose bouillon. One per cent. mannite produces about one-half of the total gas displacement in the closed branch, but, unlike dextrose, the reaction of the medium is slightly alkaline. Two parts of H to one of CO_2 is the ratio of gases produced. This group differs from the colon group in not fermenting lactose and saccharose bouillon.

The colon bacillus acidulates milk until the casein is precipitated, in from two to seven days, producing about one-sixth of total gas in the fermentation tube, with an acidity of about 6.60 per cent.

The typhoid bacillus produces from 1 to 2 per cent. acidity in milk, but no gas or coagulation is formed. The intermediate, or hog cholera, group produced about 3.6 per cent. alkali in the open bulb, as the milk is exposed to the air. In the closed bulb, from which air is, of course, excluded, about 1.60 per cent. acid is formed. No gas-formation or coagulation takes place. In all these reactions the bouillon should be carefully freed from muscle sugar according to the plan mentioned in the article on the colon bacillus.

Pathogenic Properties of the Group.—The various members of this group are very pathogenic for various animals ; as Cushing[34] points out, they may cause epidemics among hogs, cattle, mice, and birds, as well as serious outbreaks among men, such as epidemics of meat poisoning. When such animals as guinea-pigs and rabbits are inoculated with the bacillus of hog cholera, Bacillus enteritidis, or Bacillus icteroides, the animal dies from a septicemia, and focal necrosis of the liver with fatty degeneration of the organ and hemorrhages and congestion of the duodenum are found at the autopsy. The bacilli can be recovered from the blood and viscera by culture. When these animals are given nonfatal infections from one of the members of this group by intravenous injection, the blood may show an agglutinative reaction to the bacillus inoculated, to as great a dilution as 1 : 5000, and yet the animal's blood will not cause other members of the group to give a Widal reaction except in such weak dilutions as from 1 : 50 to 1 : 10, and often even this dilution fails to cause a reaction.

General Immunity.—Immunization of an animal, however, with one of these forms protects the animal against what would otherwise be a fatal dose of another member of the group. An animal which recovered from Cushing's Bacillus O survived when inoculated with a dose of the bacillus of hog cholera, which killed a control rabbit in a few hours. It will thus be seen that while immunity toward one member of the group seems to convey immunity toward other members of the group, yet an agglutinative reaction of a certain strength toward one of the members of this type does not mean that other members will react to a similar degree when added to the diluted blood of the immunized animal. Bacillus O and the paracolon bacillus are less virulent than the other members of this group.

CHIEF DISTINGUISHING FEATURES OF THE EBERTH, OR TYPHOID, GÄRTNER, OR INTERMEDIATE, AND ESCHERICH, OR COLON, GROUPS.—(*Cushing.*[34])

Bacillus Typhosus.

An actively motile bacillus possessing at times as many as fourteen flagella, and having marked pathogenicity toward man, though slight toward the lower animals. Reaction in milk is acid, with a very late (months or so) terminal alkalinity. The organism ferments glucose, but not lactose, and produces no gas from any carbohydrate medium. Under ordinary conditions of isolation there is no production of indol.

Fermentation Reactions.—

	DEXTROSE.	LACTOSE.	SACCHAROSE.
Total gas, ...	0	0	0
Aerobic, ...	Acid.	Alkaline.	Alkaline.
Reactions.—			
Anaerobic, ...	Acid.	Neutral.	Neutral.

Intermediate Group.

Bacillus Gartner and Bacillus of Hog Cholera Type.—Bacilli with the morphology of Bacillus typhosus and possessing an equal or greater number of flagella. Actively motile in many cases, having a distinct pathogenicity for both man and animals. Reaction in milk presents an early and terminal strong alkalinity in the presence of air, appearing after a transient acidity. Milk is never coagulated. They ferment glucose, with the production of gas; never lactose or saccharose. Under ordinary cultural conditions no indol is produced.

Fermentation Reactions.—

	DEXTROSE.	LACTOSE.	SACCHAROSE.
Total gas, ...	$\frac{1}{2}$	0	0
$H-CO_2$, ...	$\frac{2}{1}$	0	0
Aerobic, ...	Acid.	Alkaline.	Alkaline.
Reactions.—			
Anaerobic, ...	Acid.	Neutral.	Neutral.

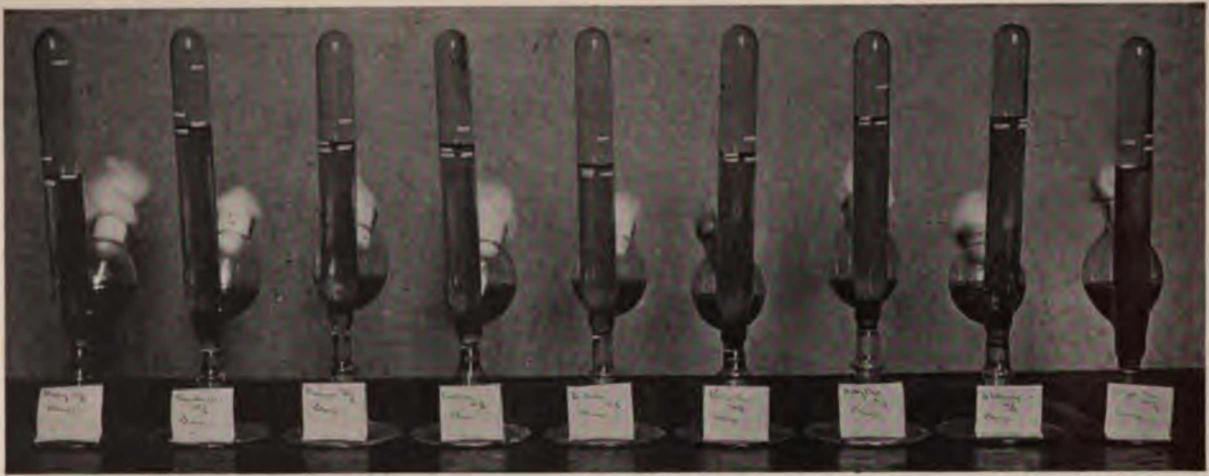


FIG. 7.—GAS-PRODUCTION IN SUGAR-FREE BOUILLON CONTAINING 1 PER CENT. DEXTROSE. THE DAILY PRODUCTION OF GAS IS INDICATED BY THE SHORT TRANSVERSE LINES. INTERMEDIATE AND COLON GROUP.—(*The five half-tones (Figs. 7–11) illustrating fermentation tests loaned through kindness of Professor Henry M. Hurd, from "Johns Hopkins Hospital Bulletin."*)

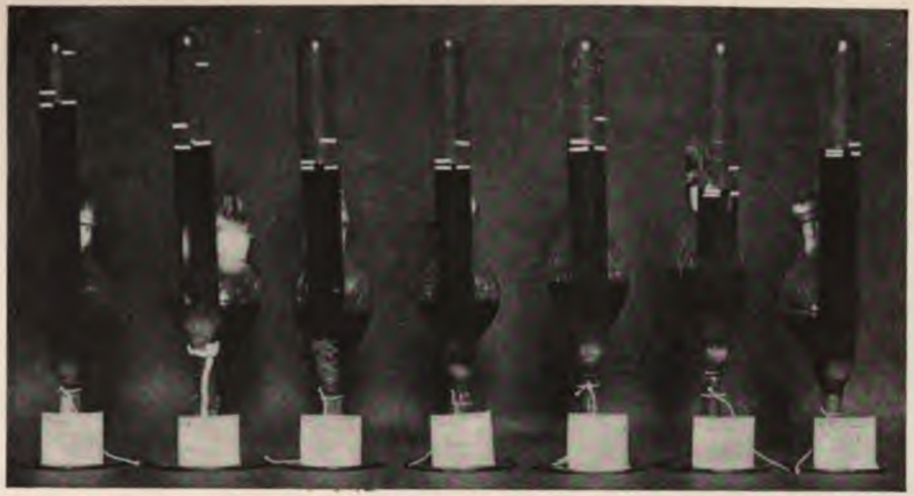
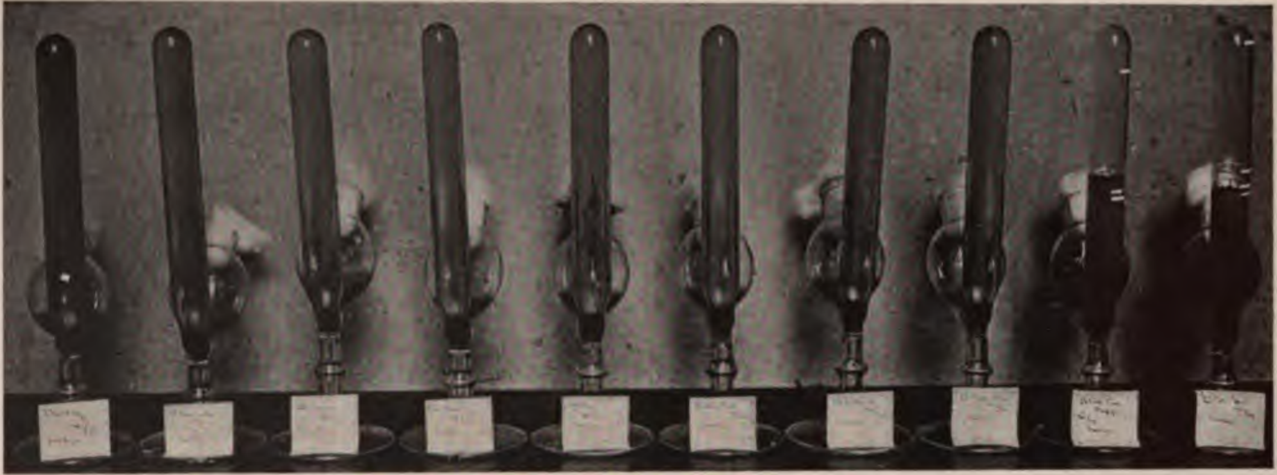
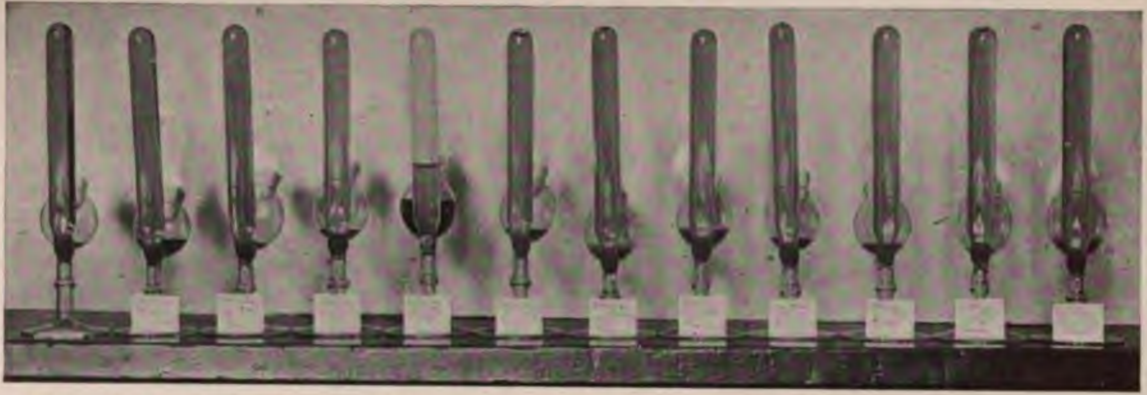


FIG. 8.—GAS-PRODUCTION OF INTERMEDIATE GROUP IN SUGAR-FREE BOUILLON CONTAINING 1 PER CENT. DEXTROSE.



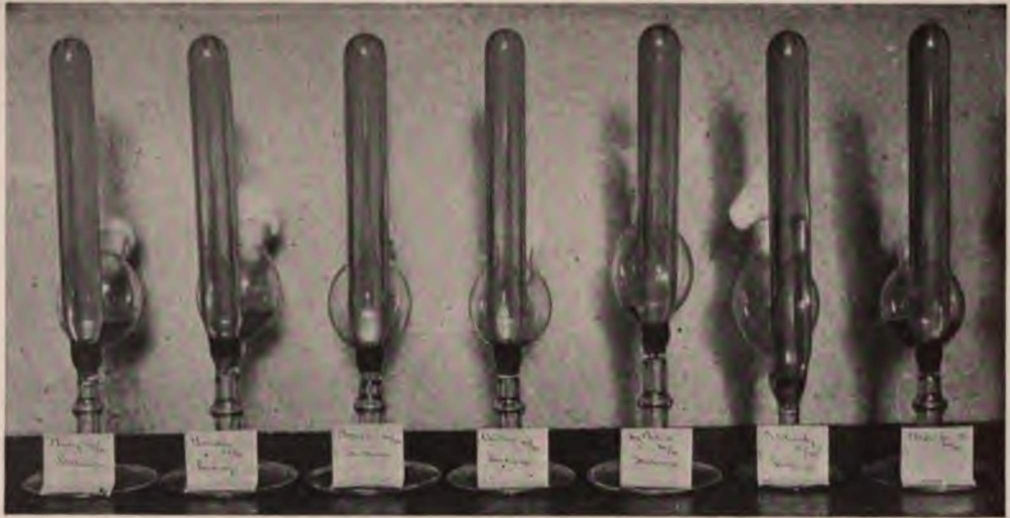
Bacillus O. | Bacillus paracolon. | Bacillus morbif. | Bacillus enteritidis. | Bacillus var. (Hatton). | Bacillus chol. suis. | Bacillus icteroides. | Bacillus typhosus. | Bacillus coli (*a*). | Bacillus coli (*b*).

FIG. 9.—TYPICAL REACTIONS OF TYPHOID, INTERMEDIATE, AND COLON GROUP IN 1 PER CENT. LACTOSE SUGAR-FREE BOUILLON.



Control. Bacillus O. Bacillus paracolon. Bacillus typhosus. Bacillus coli (*b*). Bacillus coli (*a*). Bacillus morb. Bacillus enteritidis. Bacillus Hatton. Bacillus chol. suis. Bacillus icteroides. Bacillus typhi murium.

FIG. 10.—REACTIONS OF TYPHOID, INTERMEDIATE, AND COLON GROUP IN 1 PER CENT. SACCHAROSE. BUBBLE AT TOP DUE TO SLIGHT CONVERSION OF SACCHAROSE DURING STERILIZATION.



Bacillus O. | Bacillus paracolon. | Bacillus morbif. bovis. | Bacillus typhosus. | Bacillus of hog cholera. | Bacillus icteroides. | Bacillus coli (*a*).

FIG. 11.—REACTIONS IN SUGAR-FREE BOUILLON CONTAINING 1 PER CENT. SACCHAROSE. TYPHOID, INTERMEDIATE, AND COLON GROUP.

Bacillus O and Bacillus paracolon (Gwyn) may present a subdivision of this group, being slower in their action on milk, and growing less luxuriantly and more like Bacillus typhosus in various fluid media, and resembling it in their pathogenic action on animals.

Bacillus Coli Communis and its Many Allied Forms.—A bacillus with sluggish motility, possessing few flagella. The pathogenicity ordinarily is slight for man and animals. Growth on potato is abundant and colored. No alkalinization in aerobic growth. Reaction in milk is acid, invariably sufficient for coagulation. It ordinarily produces a large amount of indol, and ferments glucose and lactose. Saccharose may or may not be acted upon.

Fermentation Reactions.—

			SACCHAROSE.	
	DEXTROSE.	LACTOSE.	Var. (*a*)	Var. (*b*)
Total gas, ...	$\frac{1}{2}$	$\frac{1}{2}$	0	$\frac{2}{3}$ slow.
$H—CO_2$, ...	$\frac{2}{1}$	$\frac{2}{1}$	0	$\frac{3}{2}$
Aerobic, ...	Acid.	Acid.	Alkaline.	Acid.
Reactions.—				
Anaerobic, ...	Acid.	Acid.	Alkaline.	Acid.

BACILLUS SUBTILIS (EHRENBERG).

Bacillus subtilis has been found both in the normal stomach- and intestinal contents.

The bacilli are in the form of long rods, about 6 μ in length, and they form oval spores. These rods are motile, and possess long flagella at each end.

Cultural Properties.—On agar a white, dry, profuse growth appears, which spreads over the entire medium. The appearance of the colonies in plate cultures of gelatin is characteristic. These appear in twenty-four hours as white points, which soon, under the low power, show a number of hair-like projections radiating from the periphery, and resembling a ray-like halo. A small ring of liquefaction soon forms around the colony, and the gelatin becomes rapidly liquefied. In gelatin stab cultures a funnel-like liquefaction begins, and later a thick membrane forms on the surface of the liquefied gelatin.

In bouillon a characteristic membrane forms on the surface and a musty smell is produced. Blood-serum is liquefied.

The growth on potato is characteristic. A profuse wrinkled membrane spreads rapidly over the surface of the medium, and this in twenty-four hours arranges itself into folds somewhat resembling a plowed field. Milk is slowly acidulated, and later coagulated. This organism is the most usual contamination of culture-media in the laboratory.

Ferments Formed.—This bacillus has no effect upon sugar, but when oxygen is supplied, it produces a ferment which rapidly digests proteids. This is easily demonstrated by its rapid liquefaction of gelatin and blood-serum. The bacillus, however, is a strict aerobic bacterium, and will not develop when oxygen is excluded.

It is thus difficult to believe that it is able to increase and develop in the intestines, since the alimentary tract is practically devoid of oxygen. Its physiological relation to digestion is, therefore, not yet proved.

The spores of Bacillus subtilis will withstand a temperature of 100° C. for one hour.

The organism is not pathogenic for animals or men.

BACILLUS BUTYRICUS (PRAZMOWSKI).

This bacillus occurs in acid and fermenting feces.

Under the microscope it appears as a long rod, from 3 to 10 μ in length, although longer rods are apparent at times. The organism is motile and forms spores.

Staining Reaction.—It possesses a characteristic staining property in taking on a violet color when cover-slip preparations are placed in solutions of iodin.

The bacillus slowly liquefies gelatin, and causes gas-formation in sugar agar and bouillon.

Cultural Properties.—Its effect upon milk is characteristic when grown at the temperature of the thermostat. In about eighteen hours the milk coagulates and is driven to the surface by the gas-bubbles which form in the medium. The fluid beneath remains clear, as the casein is dissolved in the fluid. A rancid smell is produced.

The principal acid which this bacillus forms in milk is butyric acid, but small quantities of lactic and acetic acid are also produced.

Similar products are produced in milk-sugar bouillon, and the gases produced consist of CO_2 and H. Starch is partially reduced to butyric acid by this bacillus, but cellulose and salts of lactic acid are not affected.

The organism is anaerobic and has no pathogenic properties.

BACILLUS FECALIS ALCALIGENES (PETRUSCHKY).

This organism has been found in the human feces, and resembles the typhoid bacillus in shape. It stains with the usual anilin dyes, but not by Gram's method. It is very motile, and has flagella. It does not liquefy gelatin, coagulate milk, form indol, or produce gas in sugar solutions. It will kill animals by intraperitoneal injection. It differs from the typhoid bacillus in forming spores, in alkalinizing milk, in growing visibly on potato, and by failing to give the Widal reaction with typhoid serum.

BACILLUS PUTRIFICUS COLI (BIENSTOCK).

Bacillus putrificus coli of Bienstock[10] has been found constantly in the feces and stomach-contents. It appears under the microscope as a slender rod about 3 μ in length, is quite motile, and forms spores at the end of the rod, causing the germ to appear as a large drumstick. It forms translucent, pearl-like masses on agar, and does not liquefy gelatin.

The bacillus decomposes albumins such as fibrin, forming ammonia, peptones, fatty acids, tyrosin, phenol, indol, and skatol.

The organism is aerobic, but will grow less favorably when oxygen is excluded. It is not pathogenic.

IMPERFECTLY DESCRIBED FORMS.

A number of other bacilli have been found within the intestines, but as they are usually associated with some morbid process, they will be described in another section. A few other organisms have been isolated by various authors from time to time, but as their descriptions are too meager for recognition, they are hardly worthy of notice in their present incomplete state. The confusion which exists at present concerning many intestinal bacilli and the lack of accurate knowledge make it important for future investigators to make very complete descriptions.

THE MICROCOCCI OF THE INTESTINES.

Although it was formerly taught that there were no micrococci present in the intestines, yet recent observers have demonstrated a number of cocci. These are usually found in chains, and have been described mainly by Escherich.

Intestinal Streptococci.—Streptococci coli gracilis and brevis are so similar that their characteristics can be described almost as one organism. The former is found in the intestines of adults or children who have begun to eat meat. The latter, or Streptococcus coli brevis, is found only in the feces of nursing children. Both organisms consist of cocci arranged in short chains, and they both rapidly liquefy gelatin, thus differing from Streptococcus pyogenes, which does not liquefy this medium. The gracilis coagulates and acidulates milk, and the brevis forms a citron-yellow growth on agar, blood-serum, and potato. Neither have been proved to be pathogenic. A white and a yellow staphylococcus which liquefy gelatin have also been isolated from the feces by Escherich. Cushing and Livingood[5] have found the ordinary Staphylococcus pyogenes albus in cultures from the duodenum, and they also found the Streptococcus pyogenes in cultures taken from a ruptured jejunum, in a case of jejunal fistula. The accident had occurred just after a hearty meal, with the stomach full of food.

BLASTOMYCETES, OR YEASTS.

The yeasts, or blastomycetes, do not occur in any great numbers in the intestines, but several different species have been isolated from this source.

Various Species.—A species of torula, so called by Pasteur, has been found in the meconium by Escherich. These yeast-cells often show evidence of sprouting daughter cells, but do not form ascospores or mycelium. They do not cause fermentation, and would seem to be inert organisms within the intestine. This organism will grow on gelatin or agar. The red yeast has also been found in meconium, and a large, capsulated form of blastomyces, measuring from 5 to 10 μ in diameter, has been found in feces. This yeast will liquefy gelatin. Cushing and Livingood[5] found yeast colonies in cultures from a jejunal fistula.

DIFFICULTIES OF STUDY.

From the foregoing description of the vegetable flora of the intestine it is quite evident that a great deal of systematic and comparative work must still be performed before we can claim any well-systematized knowledge of this subject. Many bacteria or other fungi will not grow on the ordinary culture-media, and often bacteria will appear in cover-slips that can not be cultivated. Special media must be devised for these organisms. Then, again, many of the germs isolated have received such superficial study that they could hardly be recognized by a second observer. But perhaps the most important and most difficult series of facts needful for the development of this subject are as yet an unsolved mystery.

Many of the intestinal bacteria form materials in culture-tubes which are also found as by-products in the intestine. Such substances are skatol, indol, various gases, etc. The point yet to be determined is by what organisms and under what circumstances the various chemical changes are brought about which are known to occur in the intestines. This subject forms a tempting field for some ambitious investigator.

THE BACTERIA WHICH ARE FOUND IN THE VARIOUS DISEASES OF THE INTESTINES.

Before considering the more special germs of the intestinal diseases, such as the Asiatic cholera spirillum and the typhoid bacillus, it might be well to mention the bacteria found in connection with the various inflammatory conditions of the intestines.

These inflammatory diseases may present a series of different clinical and pathological pictures, varying from a simple serous exudation to a condition of ulceration and fibrinopurulent deposit affecting portions of the entire intestinal tract.

A large number of organisms have been demonstrated as present either in the intestines or in the lesions. Not infrequently it has been impossible to demonstrate a direct causal relation between the disease and the bacteria.

Therefore I shall mention first the bacteria which have been simply identified as present in certain intestinal inflammations, and then describe those which have given more striking proofs as to the cause of certain conditions.

Disturbers of Function.—Bacillus butyricus, which has been previously described, has been found frequently in acid and fermenting feces, and might be considered as playing a part in the production of such a condition.

The bacillus of green diarrhea has been found by Lesage in the greenish feces of infants suffering from this form of intestinal flux. These bacilli form rods from 2 to 4 μ in length, are motile, and form spores.

The organism does not liquefy gelatin, and grows on potato and in bouillon, forming a green pigment. It is aerobic, and when introduced into the intestines of rabbits, will cause diarrhea.

The saccharomyces albicans has been found in the stools of simple diarrhea, and the writer has known of a case in which profuse watery diarrhea was apparently caused by drinking beer containing a profuse sediment of beer yeast.

Streptococcus Pyogenes.

Although the Streptococcus pyogenes has been proved by Escherich, and Cushing, and Livingood to be but an occasional inhabitant of the normal intestinal canal, yet it has recently been proved to be the cause of a severe enteritis. This condition is usually found in children and infants, but occasionally affects adults.

The streptococcus is so well described in all text-books on bacteriology that a brief description of it in this place is all that is necessary.

The organism is composed of a series of cocci growing in short or long chains, which stain by the anilin dyes and by Gram's method. It does not liquefy gelatin, and grows on agar as a series of very fine gray dots. The growth on potato is invisible, and milk is acidulated. The long chains of micrococci, especially from the water of condensation in the bottom of the tube of slanted agar or blood-serum, serve to identify it when stained and under the microscope.

It is pathogenic for animals, causing general septicemia in the mouse, guinea-pig, or rabbit.

Streptococcic Enteritis.

The streptococcic enteritis of infants, as described by Booker (*l. c.*), consists in purulent inflammation and ulceration of the mucous membrane of the intestines. Pus-cells are present in the frequent and slimy stools in great quantities, and a large number of streptococci can be demonstrated in the feces by cover-slip and cultures. These streptococci were stained by Weigert's method in the walls of the intestine and in the ulcerated mucous membrane, and they were obtained by cultures from the various internal organs, showing that the local condition often brings about a streptococcic septicemia.

Streptococci in Milk.

It is very probable that many infants are infected by drinking milk from cows with purulent inflammation of the teats and udder. Such a condition is called "garget," and the centrifugalized sediment of the cow's milk will contain many pus-cells and chains of streptococci. The milk from such cows is unfit for use.

Proteus Vulgaris (Hauser).

This organism plays an important part in certain inflammations of the intestines. It is a rather thin bacillus, and of varying length, often growing out into long threads. The organism is very motile, does not form spores, and stains by the ordinary methods, but not with Gram's stain.

This bacillus is an organism of putrefaction, and was first discovered by Hauser in decomposing meat and vegetable infusions.

The proteus grows as well at the temperature of the room (24° C.) as at that of the thermostat, and in twenty-four hours forms a profuse, gray, homogeneous growth over the surface of slanted agar. Blood-serum and gelatin are both liquefied, and indol is formed in bouillon and Dunham's solution. On potato a yellow, slimy growth is produced, and litmus milk is slowly acidulated and coagulated. The cultures all give off an unpleasant ammoniacal odor of decomposition.

It ferments sugar solutions, but differs from the colon bacillus in producing a smaller amount of gas in glucose and saccharose beef-tea, and in its inability to produce any gas in lactose bouillon in the fermentation tube. The fermentation formula of Proteus vulgaris follows :

THEOBALD SMITH'S TABLE.

SUGAR USED.	GAS PRESENT AFTER						REACTION OF BULB.	CO_2.	H.
	1 Day.	2 Days.	3 Days.	5 Days.	7 Days.	Total in 9 Days.			
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.		Per cent.	Per cent.
Glucose, ...	4	20	28	. .	35	31	Acid.	28	72
Lactose, ...	2	5	. .	8	10	10	Alkaline.	Trace.	Nearl 100
	0	0		0	. . .	. . .	Alkaline.	. . .	. .
Saccharose, . . .	6	20	30	34	36	33	Acid.	39	61
	0	24	36	32	33 (6th)	32 (6th)	Acid.	33⅓	66⅔

Chemical Products.—This organism possesses some interesting chemical properties. It produces indol and phenol from solutions of peptones, and reduces nitrates to nitrites and ammonia. In urine the proteus reduces the urea to ammonium carbonate.

The organism will cause subcutaneous abscesses when injected into the tissues of guinea-pigs and rabbits, but usually will not enter and increase in the blood. Large quantities of the bacilli will cause a toxemia if injected into the peritoneal cavity or into the blood.

Pathogenic Properties.—This bacillus has been found in human beings in such conditions as purulent peritonitis, diffuse cellulitis of the hand, pleurisy, and cystitis. As it is also the cause of several distinct diseases of the intestines, its effects in this situation will be more accurately described.

Although this organism is not a normal inhabitant of the intestine, yet Booker (*l. c.*) has shown that it is present in large numbers in the majority of cases of severe enteritis of infants and small children. These cases are usually severe, but do not show evidence of severe intestinal inflammation. The condition seems to be more of a toxemia, with the absorption of the soluble poison of Proteus vulgaris from the intestine.

Meat Poisoning.—Proteus vulgaris has been demonstrated as the cause of a disease called meat poisoning ("Fleischvergiftung"). This usually occurs from eating partially decomposed fresh meats or sausage, and spreads as an epidemic among most of the people who have eaten the infected material. The main clinical symptoms are abdominal pains, fever, and profuse serous or hemorrhagic diarrhea, and the disease may even result fatally. The proteus has been found both in the infected meat and in the intestines and feces of the patients, but it does not occur in the blood. This bears out the experimental evidence gained from animals that the organism causes an intestinal toxemia.

Silberschmidt[11] has lately recorded an epidemic of this disease, affecting forty-four persons, which was caused by sausage infected with Proteus vulgaris.

Bacillus Dysentericus (Acute Epidemic Dysentery) (Shiga and Flexner).

The cause of acute epidemic dysentery has recently been isolated from the intestinal contents, and thoroughly described by Shiga,[12] of the Tokio Institute for the Study of Infectious Diseases. This disease prevails extensively in Japan, and exists also in the Philippines.

Cultural Properties.—The bacillus consists of a short rod which stains by the ordinary dyes, but not by Gram's method. It is moderately motile, fails to liquefy gelatin, and gives an alkaline reaction to culture-media. In gelatin or agar plate cultures the colonies on the surface are bluish-white by transmitted light, round, and moist. It grows as a grayish deposit on the surface of glycerin agar and plain agar, while on potato the surface is simply rendered dry, white, and lustrous. The bacillus only clouds bouillon. The indol reaction rarely takes place in Dunham's solution, and sugar solutions are not fermented. Litmus milk is faintly acidulated in twenty-four hours, but in six days becomes alkaline and blue. No coagulation takes place.

The bacillus was obtained by Shiga in Japan by means of alkaline sugar agar from the feces in thirty-four cases of acute epidemic dysentery, and from the large flat ulcers and mesenteric lymph-glands in three autopsies. Amebæ were found only in five cases, and the bacillus was not discovered in a large number of healthy stools examined. It will grow in all the ordinary culture-media, such as agar, gelatin, potato, bouillon, and milk.

Agglutinative Reaction.—The blood-serum of twenty-five patients suffering from epidemic dysentery gave a well-marked agglutinative reaction when brought in contact with fluid cultures of the bacillus at a dilution of 1 : 10, while the macroscopic test, made by adding the serum to a beef-tea culture, caused a precipitation of the bacilli with a clear fluid above in twenty-four hours, with a dilution of from 1 : 10 to 1 : 50.

Pathogenic Properties.—If pure cultures are injected into the peritoneal cavity of a guinea-pig, the animal will die in a short time, and a hemorrhagic peritonitis is found. Young cats and dogs, if fed with the bacillus, developed a diarrhea in one or two days, and often died in four or five days. The intestines were found to be hyperemic and covered with a mucopurulent exudate, from which the bacilli were cultivated. The organs were sterile.

Shiga also performed some interesting experiments concerning immunity. He injected guinea-pigs with $\frac{1}{10}$ of a cubic centimeter of a bouillon culture of Bacillus dysentericus which had been killed by exposure to 60° C. in a water-bath for twenty minutes. On the tenth day after injection the animals' serum gave a well-marked agglutinative reaction. These interesting observations strongly suggest that Shiga has found the true cause of epidemic dysentery.

Flexner's Work.—Dr. Flexner[13] has lately confirmed these observations by a series of observations carried on in the Philippine Islands, especially around Manila. In addition to the specific bacillus of Shiga he found pyogenic cocci, Bacillus pyocyaneus, and the colon bacillus in the cultures from the intestines during life and at autopsies. Cercomonades and trichomonades were also found in the stools. None of these organisms except the specific bacillus of dysentery was considered as being concerned in the causation of the disease. He failed to find the bacillus of acute epidemic dysentery in the stools of healthy persons, and found it only rarely in the chronic or amebic form. Flexner experimented with numerous animals, with the following results. Monkeys are not susceptible, but mice and guinea-pigs will die in from one to six days when injected subcutaneously or in the peritoneal cavity, and the bacillus can be recovered by cultures from the blood and viscera.

Cats will succumb to subcutaneous injections, and dogs will develop diarrhea from feeding experiments. These animals die in five or six days, and the hyperemic and hemorrhagic mucous membrane of the intestine contains the specific bacilli. Dead cultures will also cause death in rabbits and guinea-pigs, showing that the bacillus forms a toxin.

Different from Typhoid.—It will be seen from the description of this bacillus that it is somewhat similar to the typhoid bacillus, but it differs from this organism in producing an alkaline reaction in milk, in producing indol at times, in its less active motility, and in failing to give the agglutinative test when placed in typhoid serum. Flexner also obtained the agglutinative reaction with this bacillus when it was added to the diluted blood of cases of acute epidemic dysentery, and the dead cultures will give small animals an immunity. Shiga has produced an antitoxic serum in horses by injecting vacuum-dried, twenty-four-hour agar cultures. By the therapeutic use of this serum the mortality has been reduced from about 38 per cent. to 10 per cent. Both he and Flexner agree in considering this organism as the probable cause of acute epidemic dysentery (see chapter on Dysentery) in the Philippines and Japan.

Bacillus Pyocyaneus.

Bacillus pyocyaneus is usually found in the greenish pus which often discolors the dressings of an open wound. It is also found as a saprophyte in the axilla, auditory canal, and intestinal mucus.

It is a slender bacillus, measuring from 1 to 2 μ in length, and possessing many flagella. It stains by the ordinary anilin dyes, but not by Gram's method, and it has no spores. It is actively motile, and practically an aerobic organism. It produces an acid reaction.

Cultural Properties.—When grown on agar, a light green smear is found, and this green color is imparted to the entire medium in forty-eight hours. The medium later turns very dark green. On potato, a yellowish-brown growth is formed, which will turn green when the superficial portion is removed by scraping. It soon again resumes its brown color, and this characteristic reaction is called the "chameleon phenomenon."

Gelatin is rapidly liquefied, and under the low power the deep plate colonies are coarsely granular, with irregular filamentous borders. In stab cultures a funnel-shaped area of liquefaction forms, which gradually extends until the fluid and solid portions of the medium are separated by a horizontal plane. The greenish color quickly extends through the entire medium. Milk is acidulated and coagulated, but the casein is later peptonized, precipitated, and slowly redissolved, the fat rising to the top. The bacillus does not cause any fermentation.

The green pigment is due to the formation of pyocyanin, a material which is soluble in chloroform and which crystallizes in the form of long blue needles.

The organism is pathogenic for guinea-pigs and rabbits, and when one cubic centimeter of a bouillon culture is injected into the circulation or peritoneal cavity, the animal dies in twenty-four hours. At the autopsy the bacilli are present in the blood and various organs, or in the fibrinopurulent exudation in the abdominal cavity. When subcutaneous injections are made, the bacilli are found in the edema or purulent infiltration of the tissues.

Bacillus pyocyaneus was first observed in the green pus of open wounds, and later in otitis media, brònchopneumonia, abscess of the liver, inflammation of the pericardial sac and knee-joint, general septicemia, and hematemesis of the new-born. It has also been found by Lartigau[35] in the stools and intestines of a number of cases of serious and even fatal epidemic dysentery, and in one epidemic the drinking-water from the surrounding wells also contained the organism.

Bacillus Botulinus.

Proteus vulgaris has already been mentioned in describing meat poison, but Bacillus botulinus must also be mentioned in this connection.

This organism was isolated by Van Ermengen in an epidemic of sausage poisoning, and has the following characteristics:

The bacillus is a large, slightly motile rod, from 4 to 6 μ long, with rounded ends and flagella. Spores are formed in the body and also in the cultures, and the bacillus stains with the ordinary anilin dyes and by Gram's method.

It grows best at a temperature of from 20° to 30° C., and is strictly anaerobic. The colonies in gelatin plates appear about the fourth day as round, transparent, brownish-yellow masses, surrounded by a slight area of liquefaction. A whitish growth takes place on agar, and bouillon is rendered turbid. Milk and potato are not affected. In glucose bouillon the organism produces gas.

The bacillus remains alive for one year if kept at 30° C., but the spores and bacilli are destroyed by a temperature of 80° C. in one hour.

Pathogenic Properties.—Bacillus botulinus kills guinea-pigs, mice, and monkeys when placed in their food, the animals dying with paretic symptoms, and subcutaneous injections will also produce this result. The bacillus is found in the blood and internal viscera if the animal is allowed to remain at a temperature of 30° C. for twelve hours.

A toxin has also been secured by filtering fluid cultures, which will cause the same effect as the bacillus if injected into the tissues.

Bacillus botulinus was found in a ham and in the body of a patient who had died of botulism. They were obtained in cultures from the spleen, the stomach, and the intestines. It is probable that this organism causes many of the cases of meat poisoning, and when searching for it, the fact should be borne in mind that it is a strictly anaerobic organism.

The Bacillus of Typhoid Fever (Eberth).

Bacillus typhosus, or the typhoid bacillus, is present in the feces, urine, and less frequently in the blood and internal organs of persons having typhoid fever.

This bacillus was first described by Eberth in 1880, who demonstrated it in stained sections of the organs of typhoid cases, but it was not secured in pure culture until 1884 by Gaffky.

Morphology.—The germ is a bacillus with rounded ends, and is from 1 to 3 μ in length, although it frequently grows out into very long rods. These possess numerous flagella, and divide by transverse fission. The bacillus stains by ordinary dyes, at times irregularly, but it is decolorized by Gram's method. It is actively motile, especially in cultures from twelve to twenty-four hours old. This motility is caused by a number of fine, hair-like processes from either side of the rod. They usually number about twenty, and require special stains for their demonstration.

Although there have been numerous methods devised for staining flagella, the stain proposed by Löffler is perfectly satisfactory.

Stain for Flagella.—The cover-slips used should be perfectly new, as those which have been used contain an oily matter which prevents proper spreading. The slips must be carefully cleaned in alcohol, and wiped dry just before using them. A small amount of an eighteen-hour culture on glycerin agar is placed in a drop of water on the first slip, and a small drop is removed with a loop, and very thinly spread over a number of other slips which have already received a small drop of water. The cover-slips must be dried in the air and passed through the flame three times. The following mordant is then placed on the cover-slip for a minute:

Twenty per cent. solution of tannin,	10 c.c.
Cold saturated solution of ferrous sulphate,	5 "
Alcoholic solution of fuchsin,	1 "
One per cent. caustic soda solution,	1 "

The freshly prepared mordant should be slightly warmed over a flame, but *not* boiled. It is then washed off in water, and the preparation is stained with Ziehl's carbolfuchsin solution, washed in water, dried, and mounted in balsam. This is a difficult stain to manipulate, and numerous failures will occur, but at times beautiful demonstrations of the flagella will be obtained. Other stains, such as Van Ermengen's and Pitfield's flagella stain, can be found in any of the recent text-books on bacteriology.

Cultural Characteristics.—The organism grows well at room-temperature, but better at 35° C., and is greatly limited in growth by 40° C. The thermal death-point is 56° C. for ten minutes. Sternberg has kept hermetically sealed bouillon cultures alive for one year, and freezing does not destroy the organism. It remains alive for three months in distilled water, and for six months in soil. It resists drying for ten weeks. A solution of 1 : 10,000 of bichlorid of mercury will destroy the typhoid bacillus in two hours, but if egg-albumen is added to a bouillon culture, it takes a 1 per cent. solution of mercuric chlorid to destroy it, since the albumin forms an insoluble vehicle. This should be borne in mind in disinfecting feces and urine. A 1 : 200 carbolic and a 1 : 1000 formalin solution will destroy this organism.

The typhoid bacillus is an aerobic organism, but will grow less favorably when oxygen is excluded.

The organism grows well on all the usual culture-media. On the surface of slanted agar, blood-serum, and gelatin it forms a profuse, grayish, moist growth, and isolated colonies are of a bluish, pearl-colored appearance when viewed by transmitted light. The deep colonies show no characteristic appearances in ordinary plate cultures, and do not liquefy gelatin. Bouillon and Dunham's solution are clouded, but no surface membrane is formed, and indol is not produced. Litmus milk usually remains unchanged for about one week, and then an acid is slowly formed, but some varieties form this acid and redden the medium in twenty-four hours. It later becomes alkaline. An invisible growth is formed on potato. The organism does not produce any gaseous fermentation in glucose, lactose, or saccharose bouillon in the fermentation tube, and this invariable characteristic is its most important reaction in distinguishing it from Bacillus coli communis. If fermentation tubes are not at hand, the bacillus can be sowed in 1 per cent. melted glucose agar cooled to 40° C., and then allowed to solidify and develop in the thermostat for twenty-four hours. The typhoid bacillus will not form any gas-bubbles, but the colon bacillus ferments the glucose and forms numerous gas-bubbles in the solid agar.

DIFFERENTIAL DIAGNOSIS BETWEEN THE TYPHOID BACILLUS AND BACILLUS COLI COMMUNIS.

Bacillus Coli Communis.	Bacillus Typhosus.
Coagulates and acidulates milk.	No effect on milk.
Luxuriant, moist, waxy growth on potato.	Invisible growth on potato.
Sluggishly motile or inactive.	Actively motile.
Indol reaction in Dunham's solution.	No indol reaction.
Gas-production in sugar solutions.	No gas-formation.
Does not react with typhoid blood.	Gives the Widal reaction with diluted dried typhoid blood or serum.
Large colonies in Elsner's or Hiss' medium.	Small characteristic colonies in Elsner's and Hiss' medium.

Pathogenesis.—Typhoid fever has never been exactly reproduced in the lower animals, although typhoid feces have been fed to such animals as swine, apes, dogs, cats, rabbits, guinea-pigs, and white mice. Pure cultures of the organism have also been introduced by Gaffky into the intestines of apes, rabbits, guinea-pigs, white rats, mice, pigeons, chickens, and calves. All these attempts to produce the fever and intestinal lesions of the human disease were negative. But although this is the case, yet if from one to three cubic centimeters of a pure culture of the typhoid bacillus are injected into the peritoneal cavity of a guinea-pig, the animal will die, and the organism will be found in the blood and internal organs. If the dose is not sufficient to produce a fatal result, the animal after recovery is immune to the typhoid bacillus. A guinea-pig can also be immunized by gradually increasing doses of dead typhoid bacilli. The bacilli are killed by heating a slanted blood-serum culture for one hour at a temperature of 60° C. About two milligrams of the pasty mass are suspended in sterile water and injected into the peritoneal cavity of the guinea-pig. This dose can be repeated in two days, and the animal is then immunized.

The blood of these animals shows a characteristic reaction, which was first described by Pfeiffer, and it also contains a protective substance granting immunity against further infection by the typhoid bacillus.

The Pfeiffer Reaction.—The Pfeiffer reaction is produced by collecting some blood-serum from the immunized guinea-pig and diluting it 100 times with sterile water. A few motile typhoid bacilli are then added, and the mixture is injected into the peritoneal cavity of nonimmunized guinea-pigs. If minute portions of this fluid are withdrawn from the peritoneal cavity from time to time, the motility will be seen to have ceased, and the bacilli first clump in small masses, then swell up, become pale, and finally dissolve in the fluid. This phenomenon usually takes place in about twenty minutes, but it will not occur if the serum of normal non-immunized guinea-pigs is diluted and injected. It is thus a specific reaction, and animals immunized against other bacteria, such as the spirillum of Asiatic cholera or Bacillus coli communis, will give this reaction in their serum to the organism which was used for immunizing the animal, but will fail to give the reaction when any other organism is mixed with their diluted serum.

The Widal Reaction for Typhoid Fever.—Although the

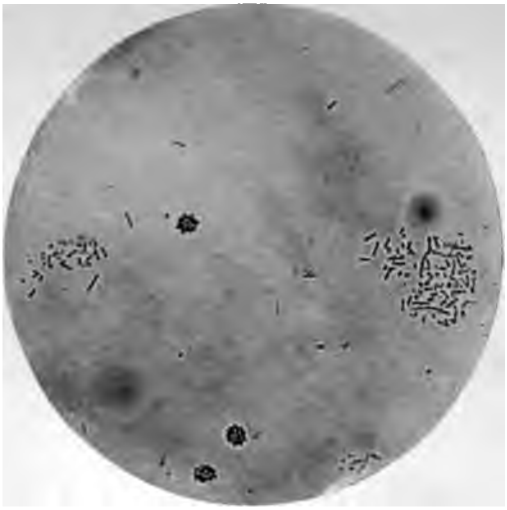


Fig. 12.—A Positive Typhoid Reaction. Typhoid Bacilli in a Bouillon Culture with which Typhoid Diluted Blood-serum has been Mixed.

The characteristic clumping of the bacilli is shown; also three crenated red blood-corpuscles.—(*Wright and Brown, Clinicopathological Laboratory, Massachusetts General Hospital. Used by permission of authors.*)

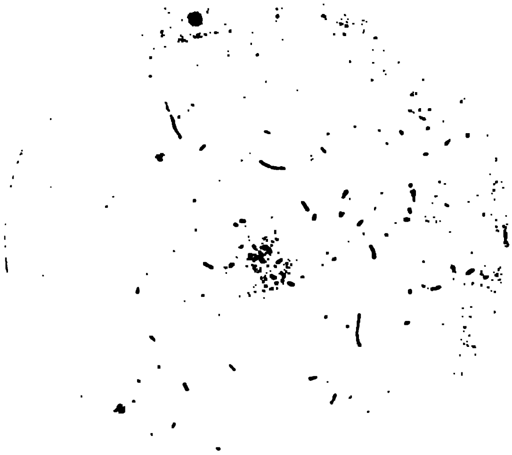


Fig. 13.—A Negative Typhoid Reaction. Typhoid Bacilli in a Bouillon Culture, as seen under a High Magnifying Power, Before the Addition of Typhoid Blood-serum.—(*Wright and Brown, Clinicopathological Laboratory, Massachusetts General Hospital.*)

work of Pfeiffer has formed the basis of all further investigations concerning the various problems bearing on immunity in typhoid fever, yet in addition it has served to develop a clinical test of great importance to physicians. This is called the Widal test for typhoid fever, and it was the direct outcome of Pfeiffer's experiments.

This experimenter soon found that his phenomenon would occur if the serum of immune animals was simply properly diluted in a twenty-four-hour bouillon culture of the typhoid bacillus. The addition of the serum to bouillon also caused a striking macroscopic test. If the serum of the immune guinea-pig was diluted with forty parts of a bouillon culture of the typhoid bacillus, and this same dilution was made with the serum of normal or nonimmunized pigs, the two tubes would soon show a striking contrast. If the tubes are placed in the incubator at 37° C. for four hours, the tube containing the serum of the immunized pig will contain an obvious sediment of agglutinated typhoid bacilli, while the supernatant fluid will remain perfectly clear. The tube of bouillon containing the nontyphoid serum from the nonimmunized guinea-pig will not contain any sediment of clumped typhoid bacilli, but will be universally clouded by the growth of the typhoid bacillus. This test will remain apparent only for about twenty-four hours.

Macroscopic Test for Typhoid Fever.—This method will serve as a test for typhoid fever, and the blood of typhoid patients will often give the so-called macroscopic reaction.

It is secured by adding one part of blood or serum to ten parts of a twenty-four-hour culture of the typhoid bacillus, and placing it in the incubator. In from four to twenty-four hours the agglutinated bacilli will form a sediment in the bottom of the tube, and the fluid above will remain clear. This method is not so reliable as the microscopic test, but it can be used when no microscope is available.

Microscopic Tests.—The practical utility of the microscopic test in diagnosticating typhoid fever was first emphasized by Widal, and to him we owe the serum method. This is performed in the following way:

The blood is secured from the lobe of the ear after washing the lobe. The puncture is made by a bayonet-pointed surgical needle or a special puncture knife.

Fifteen drops of blood are caught in a 2-inch test-tube, from which about three drops of serum will separate. In about five minutes the clot separates from the side of the tube, and the serum is ready for testing.

Forty drops of a twenty-four-hour bouillon culture of the typhoid bacillus are well mixed with one drop of the serum with a medicine dropper in a test-tube, and a loopful of this mixture is placed on a hollow glass slide. If the case is one of typhoid fever, the bacilli at some time within the space of an hour will clump into large masses, with no bacilli in the spaces between the clumps, and the motility of the bacilli will cease. If the disease is not typhoid fever, the motility will continue, and the agglutination or clumping will not take place. If no reaction takes place with a dilution of 1 : 40, it is better to make a second dilution of 1 : 20, and observe it for one hour. If these tests are not positive, they should be repeated from day to day until the reaction occurs or all suspicion of typhoid fever has passed. In the very early stages of typhoid fever the reaction will occur only in a dilution of one part of serum to ten parts of the culture, but the reaction should occur in fifteen minutes in order to be reliable. In certain cases of typhoid fever the reaction occurs at once, but it often is not complete until the very end of the time limits previously mentioned. The dilutions of serum and culture can be conveniently made in an ordinary blood counter. Although the average strength of dilution in which the typhoid reaction will occur is 1 : 50, yet 1 : 500 is not rare, and an agglutinative power has been observed when the serum was diluted 1 : 12,000 times.

The Test with Dry Blood.—Wyatt Johnston, of Montreal, was the first person who really placed the test for typhoid fever within the possession of the practising physician as a valuable clinical sign. This investigator demonstrated the fact that the dried blood of a typhoid patient will give the typhoid reaction even when considerably diluted with distilled water or bouillon. This discovery became of immediate importance, and is now used in every laboratory as a routine test.

The simplest method of applying the test is to have the physician collect about five drops of blood on a piece of nonabsorbent paper. The speck of blood can be diluted about five times, and an equal amount of a twenty-four-hour motile bouillon culture of the typhoid bacillus is then added to the diluted blood on a cover-slip. This makes a dilution of 1 : 10. If the reaction does not take place in fifteen minutes, the test should be considered negative.

A more exact way of applying this test is obtained by collecting the blood in the eye of a platinum or wire loop two millimeters in diameter. The blood from the loop is then deposited on a paper or glass slide, and allowed to dry. When the blood arrives at the laboratory, ten drops of water can be added to one of the drops of dry blood with a loop of the same diameter, and a loopful of this diluted blood added to the same loopful of a bouillon culture will make a dilution of 1 : 20. This should be observed for one hour for agglutination and cessation of motility before making a negative report. Dilutions of any strength can obviously be secured by this method.

The most accurate method for using dried blood is to weigh out one milligram of dried blood. In order to secure these dried scales of blood the physician can be instructed to collect the blood in aluminium boxes, which cause the blood to scale off when dry, and form very convenient particles for weighing. This method, proposed by Westbrook, has been used extensively for examining the dried scales of blood sent by physicians to be tested for the typhoid reaction. This consists in adding one milligram of dried blood to 100 mg. of distilled water. The blood is allowed to dissolve thoroughly in the water for one hour, and one loopful of a twenty-four-hour fluid bouillon culture is added to one loopful of the dissolved blood on a cover-slip. This is an apparent dilution of 1 : 200, but is in reality only 1 : 50, since dried blood loses three-quarters of its original weight as a fluid.

The bouillon is made exactly neutral to phenolphthalein. The cultures are allowed to develop at the ordinary room-temperature. The blood is collected by the physicians in aluminium boxes and can be sent by mail. The practitioners are carefully instructed to obtain large enough drops to weigh, as they easily scale off the metal after drying. Very often, however, they secure such small flat drops that the less accurate plan of making a dilution of 1 : 20 by the eye has to be adopted. The specimen should be observed for two hours before making a negative report.

Technic.—There are a few points concerning technic which should be mentioned in describing this reaction. In the first place, the relation of the *time limit* for observing the test before making a negative report to the dilution of the blood is important. Often pure *normal* blood will cause clumping of the typhoid bacillus. The typhoid reaction is *quantitative*, not *qualitative*. As Cabot,[14] in his excellent book, expresses it, "Typhoid serum differs from normal serum in that it will clump typhoid bacilli in a shorter time and in greater dilution than will normal serum or that of other disease." This book is one of the best treatises on this subject in print. A dilution of 1 : 50 should be observed for two hours; 1 : 40 and 1 : 20 for one hour; while if a dilution of 1 : 10 does not clump in fifteen minutes, the reaction can be considered negative.

Culture Used.—The culture used for the typhoid test should be a twenty-four-hour motile growth, preferably in neutral bouillon. If the culture is allowed to grow in the incubator at 35° C., it will often contain small clumps. These are misleading, and at times give rise to pseudo-reactions which might cause a mistake in diagnosis. In order to avoid these artificial clumpings in the culture, various expedients have been devised by different authors. Johnston avoids pseudo-clumping by transplanting one-month-old agar cultures into the bouillon, and making the bouillon a 3.5 per cent. acid. This can also be avoided by using perfectly neutral bouillon, and allowing the culture to develop at room-temperature.

Cabot has collected the records of 5978 clinical cases of typhoid fever, and he found that a positive blood-test was obtained in 5814 cases, or 97.2 per cent. In 6668 cases of other diseases the test was said to have been obtained in 5 per cent. of cases, but Cabot considers that most of them were probably masked typhoid cases or imperfect reactions.

The reaction will not occur usually until the fifth day of fever, and may be delayed much longer, so that when negative reactions are obtained, the test should be repeated until positive, or until all suspicion of typhoid fever has passed. Out of 849 cases tested, the reaction occurred before the eighth day in 93 per cent. of cases.

The Widal test for typhoid fever may be considered as a specific reaction, present only in the blood of a typhoid patient, and not characteristic of any other disease.

Human Pathology.—Typhoid fever is produced in the human being by the introduction of the typhoid bacillus into the stomach and intestines by means of fluid or solid food. It is a typical water-borne disease, and is thus most frequently caused by infected water. This is proved by the reduction of the typhoid mortality from fifty to ten in 100,000 inhabitants when the water-supply is purified by sand filtration or by the use of artesian wells and mountain springs. A number of interesting typhoid epidemics[15] have been traced to the introduction of sewage or nondisinfected typhoid feces into the intake pipes or reservoirs supplying various communities. The final proof has lately been added by Hankin[16] and Kuber,[17] who have demonstrated typhoid bacilli, characteristic in all respects, even to giving the Widal reaction in several piped water-supplies during a typhoid epidemic and in a spring.

Epidemics following the route of a certain milkman with walking typhoid, local outbreaks from oysters culled at the outlet of a sewer, and the frequent attacks resulting from the nondisinfected hands of attendants upon typhoid fever are also examples of infection by means of the alimentary tract. The report of the committee appointed by Surgeon-General Sternberg[18] shows that the flies may transport the typhoid bacilli from open latrines containing feces to various articles of food. This is done by means of the soiled moist feet or wings of the flies, and was said to cause much of the typhoid fever in the army camps during the late Spanish-American war. In many instances the kitchens or even the eating tents were close to the latrine, and swarms of flies transported the moist fecal material to the bread, fluids, and other articles eaten by the soldiers. It has been proved experimentally that flies can transport the germ on their wings and feet.

Typhotoxin.—The chemical formula of typhotoxin is $C_7H_{17}NO_2$, and it is obtained by cultivating the bacillus for two weeks in bouillon, and then extracting the base by mercuric chlorid and hydrogen sulphid. This substance is poisonous to guinea-pigs in doses of about 0.3 gm., causing paralysis, convulsions, lowering of temperature, diarrhea, and death. Typhoid fever is not frequently a septicemia, and many of the symptoms, as fever and the various nervous symptoms, are probably caused by the absorption of this typhotoxin after it has been produced by the typhoid bacillus in the intestines.

This toxin not only causes these effects, but it is thought by Mallory[19] that it produces a proliferation of the endothelial cells in the lymphatic apparatus, and even in the blood-vessels of the intestines. These cells are large, with large, lightly staining nuclei, and they assume a phagocytic property. They are later produced in the various other lymphatics and lymph-spaces of the body. This phenomenon causes the well-known swelling of the lymphoid elements of the intestine, and the secondary necrosis and ulceration of Peyer's patches and solitary follicles are due to the closure of the small veins and capillaries by these phagocytic cells and the resulting fibrinous thrombi. The focal lesions of the liver are also due either to the plugging of the capillaries of the liver by these large cells or to the proliferation of cells from either the capillaries or lymph-spaces of the liver. Focal lesions may also form in the kidney, heart, and testicles, owing to the same causes. The lesions of typhoid fever are proliferative, but they are diffuse and bear more of a relation to the toxin than to the bacillus.

Isolation from Feces.—It is very difficult to isolate the typhoid bacillus from the intestines by means of an ordinary agar plate culture, and various selective media have been devised. Perhaps the most satisfactory of these methods is that proposed by Hiss,[20] since the medium is semisolid and can be examined after twenty-four hours in the incubator.

This medium is composed of ten grams of agar, twenty-five grams of gelatin, five grams of salt, ten grams of glucose, and five grams of Liebig's beef-extract to a liter of water. The glucose is added after clearing, and the reaction should contain 2 per cent. of normal HCl, phenolphthalein being used as an indicator. The usual precautions in making up culture material should be used.

After twenty-four hours the typhoid colonies can easily be distinguished from the colon colonies under the low power. The deep colon colonies are much larger than the typhoid colonies, and are opaque, spherical, or whetstone-shaped, and brownish-yellow.

The typhoid colonies are much smaller and quite characteristic. They are small, greenish yellow, and contain a number of filamentous outgrowths, forming a complete fringe around the periphery of the colony. This fringe growing at right angles to the periphery is the important point for recognition.

In order to isolate the typhoid bacillus from feces, a platinum loopful of feces should be thoroughly suspended in 10 c.c. of sterile water. The first plate culture should be prepared from this suspension, and a second plate from the first tube of Hiss' medium, since the first plate may be too crowded. Characteristic fringed colonies can be picked out on the end of a platinum needle, and pure agar slant cultures can be subjected to the fermentation and other cultural tests for typhoid. The bacillus can even be tested for the Widal reaction with known typhoid blood if the identification is very important.

Elsner's[22] potato gelatin and Piorkowski's[23] urine gelatin are also used to separate the typhoid from the colon bacillus in feces.

Richardson[24] has lately demonstrated the typhoid bacillus in 44 out of 172 tests made for its presence in the urine of typhoid patients. The bacilli appear in the later stages of the disease and persist during convalescence. These findings have been confirmed by others, and the bacillus has also been found in cystitis and cholecystitis, at times a year or more after typhoid fever.

THE SPIRILLUM OF ASIATIC CHOLERA (KOCH).

The spirillum of Asiatic cholera was discovered by Robert Koch in 1883, who found the germ present in large numbers in the intestines, but not present in the internal organs. The spirillum was found in the feces, especially the mucous flakes of 100 cases, and was stained by the anilin dyes and cultivated in gelatin.

Morphology.—The organism consists of slightly curved rods with rounded ends, and they are often called comma bacilli, and in old cultures these rods grow out into long spiral forms with a few irregular involution types. No spores are formed, but many terminal flagella can be demonstrated by Löffler's stain. These flagella are stained exactly as the typhoid flagella, excepting the use of an acid instead of an alkali. One drop of a 1 per cent. solution of H_2SO_4 is added to the mordant.

The spirillum stains by the ordinary anilin dyes, but not by Gram's method, and twenty-four-hour cultures are actively motile.

The thermal death-point of the organism is 52° C. for four minutes. When moist, it will remain alive for nine months, but when dried on a cover-slip, it is destroyed in three hours. It can be destroyed by a 1 : 10,000 solution of bichlorid of mercury. A good practical disinfectant for the hands or even the stools is a 1 : 1000 solution of bichlorid of mercury, or a solution of similar strength of formalin.

Cultural Characteristics.—The cultural characteristics are of great aid in distinguishing the germ of Asiatic cholera, and the growth in gelatin is characteristic. In gelatin plate cultures grown at room-temperature (about 22° C.) a number of fine dots appear in twenty-four hours, which, under the low power, appear pale and finely granular, this condition becoming more pronounced, and in forty-eight hours the coarsely granular appearance is said to resemble broken glass. The liquefaction of the gelatin soon forms a halo around the colony, which in another twenty-four hours consists of a series of concentric rings or zones.

In a gelatin stab culture the appearance is also characteristic. In from twenty-four to forty-eight hours a grayish growth appears along the line of puncture of the gelatin, and a small point of liquefaction appears on the surface of the gelatin. This increases in size until it resembles an air-bubble, with a sediment in the bottom consisting of the bacteria. This bubble gradually assumes a funnel shape, and in two weeks the upper two-thirds of the medium is liquefied.

On agar the spirillum forms a grayish-yellow growth, and on potato a yellowish-brown growth is produced. In bouillon and Dunham's solution the germ grows very luxuriantly, and forms a characteristic pellicle or scum-like membrane on the surface. In milk the medium is slowly rendered acid.

On adding five drops of pure sulphuric acid to a twenty-four-hour culture of the spirillum of Asiatic cholera in Dunham's solution, the so-called cholera red reaction is produced. This is due to indol, and immediately forms a reddish-pink reaction. This reaction is very important in distinguishing it from the spirillum of Finkler-Prior, which does not give any indol reaction in twenty-four hours. Solidified blood-serum is liquefied by the cholera spirillum.

Rapid Method for Diagnosis.—Schottelius has shown that the spirillum will develop more rapidly than other bacteria when placed in diluted bouillon. By adding one part of bouillon to five parts of sterile water and then inoculating it with cholera dejecta, he found that the cholera germs will outgrow the colon bacilli and other bacteria present, and form a pellicle on the surface in twenty-four hours. This membrane will almost entirely consist of the cholera spirillum, and pure cultures can thus be easily obtained. This forms a good rapid method for the diagnosis of cholera.

No fermentation is produced in the various sugar solutions.

Pathogenesis.—Numerous attempts have been made to produce cholera in various animals. They have been fed with the dejecta and the vomit of cholera patients time and again, but with negative results. Pure cultures have also been given with negative result. No animals have been infected with cholera successfully in a perfectly natural way, and the disease is characteristic only in human beings. Koch, however, has produced in an artificial manner the infection in guinea-pigs by first neutralizing the contents of the stomach with five cubic centimeters of a 5 per cent. solution of carbonate of soda, injected into the stomach through a small catheter. One cubic centimeter of tincture of opium for every 200 gm. of guinea-pig was injected into the peritoneal cavity to control the peristaltic movements, and then ten cubic centimeters containing a few drops of the cholera culture were introduced into the stomach through the same catheter. The animals soon became weak and paralyzed, and death occurred with collapse and fall of temperature in about forty-eight hours. At the autopsy the small intestine was congested and filled with a watery fluid containing many spirilla.

Accidental Human Infection.—Several cases of accidental infection of human beings have occurred, showing positively that the cholera bacillus will produce the disease in men. In 1884 one of Koch's students became ill with cholera while experimenting with this organism in the laboratory; and in 1892 Pettenkofer and Emmerich intentionally swallowed fresh cholera cultures in order to prove a certain pet theory. Pettenkofer had a mild attack of cholerine, but Emmerich became very ill, with characteristic symptoms of cholera. Dr. Oergel, of Hamburg, and a student in Paris also became accidentally infected with cholera.

Thomas,[25] Issaeff, and Rolle[26] have produced death in young rabbits by injecting fluid cultures into the ear vein.

Immunity Against Cholera.—Koch and Pfeiffer found that small portions of agar cultures when injected into the peritoneal cavity would usually cause the death of the animals; but if the animals recovered, they were immune to very virulent cultures. R. Pfeiffer also discovered that he could produce an acquired immunity in guinea-pigs in the following manner: He first destroyed a slant culture by 70° C., and then injected two milligrams of these dead cholera germs into the peritoneal cavity of a guinea-pig. This dose was repeated after about two days, and the animals were then immune to cholera. Their blood-serum would always give the Pfeiffer reaction, and this was obtained in the following way:

One part of blood-serum was added to 100 parts of a bouillon culture of cholera, and one cubic centimeter is injected into the peritoneal cavity of young guinea-pigs weighing 200 gm. The peritoneal fluid is withdrawn by means of capillary tubes every few minutes, and when examined under the microscope, the cholera spirilla become motionless, swell up, and at last dissolve in the serum. This takes place in about twenty minutes, and is due to the formation of the so-called "Cholera-Antikörper." This phenomenon will also take place in the test-tube if one part of the serum of an immune guinea-pig is added to 100 parts of the bouillon culture.

This Pfeiffer reaction formed the groundwork of all the important work which has been performed in the immunity produced in similar diseases, and the Widal test for typhoid fever was the outcome of this research.

Widal Reaction in Cholera.—After the first twenty-four hours of cholera in human beings the blood and serum will give a modified Pfeiffer reaction, as shown by agglutination and cessation of motility when diluted from 1 : 10 to 1 : 20. This has been used with success as a valuable clinical test in cholera.

A poisonous proteid is formed in cultures of the cholera spirillum, called toxopeptone, because it resembles a peptone in its chemical properties. It produces paralysis and death in doses of about 0.1 gm. in guinea-pigs, also producing injection of the intestines and hemorrhages. Putrescin, a ptomain with the formula $C_4H_{12}N_2$, has also been isolated from fluid cultures of the organism.

Cholera in human beings is a toxemia, as the spirillum is found only in the intestines and not in the internal organs. In this situation it elaborates its poisons, and their absorption into the circulation causes the typical symptoms of the disease.

Spirillum of Finkler-Prior.

This organism has been isolated by Finkler and Prior from a number of cases of cholera nostras, or pseudo-cholera, and is probably at times a harmless inhabitant of the human intestine. It is important, however, in its resemblance to the spirillum of Asiatic cholera. At times it might be a fatal mistake to confuse one organism with the other, and their differential diagnosis is therefore given.

Differential Diagnosis.—The spirillum of Finkler-Prior is said to be somewhat longer and thicker than the organism of true cholera. Like true cholera, it is a motile organism which liquefies gelatin and blood-serum, and forms a grayish-yellow slimy growth on potato. When injected into the intestines or peritoneal cavity of animals, it acts in the same manner as does Asiatic cholera, and animals immunized to the organism will show Pfeiffer's reaction.

The important cultural test which serves to distinguish it from the spirillum of Asiatic cholera is its failure to produce indol in a twenty-four-hour growth in Dunham's solution. When five drops of H_2SO_4 are added to such a culture, no reaction takes place, but when this acid is added to Asiatic cholera, the characteristic cholera red reaction appears at once. This is the most important test in distinguishing the Finkler-Prior spirillum from that of Asiatic cholera.

Bacillus Aërogenes Capsulatus (Welch); Bacillus Welchii (Migula).

This organism has assumed considerable importance, and as it has been found to be a frequent inhabitant of the intestines of men and animals, and to be widely distributed in the outer world, it will be described at some length in this article.

Bacillus aërogenes capsulatus was discovered by Welch[26] in 1891, and has since been found by numerous independent observers. Its main characters were fully described by Welch and Nuttall and Welch and Flexner in 1892 and 1896.

Morphology.—The bacillus is large, and measures from 3 to

5 μ in length. It is about the size of the anthrax bacillus, and has slightly round or square ends. It may grow in short chains, but rarely in filaments. It stains with all the ordinary stains and by Gram's method, and can be demonstrated in tissues by Gram's or Weigert's bacterial stains. A capsule can usually be stained in specimens from blood, and sometimes in cultures, by placing a few drops of strong acetic acid on the dried and heated film, and then, after letting this run off, a strong aqueous solution of gentian-violet is at once poured on. The specimen is examined in the staining fluid, the excess of fluid being blotted off the slide. This shows stained capsules better than specimens mounted in balsam.

The formation of spores is inconstant, depending on the culture-medium and other circumstances. They often appear in cultures on Löffler's blood-serum, alkaline starch agar, and in mannite bouillon, sometimes also on plain agar, very rarely in the fresh blood and tissues of animals. They resist drying for at least a year, and can be stained by hot solutions of fuchsin in anilin water. The bacillus is not motile.

Cultural Properties.—The gas bacillus is strictly anaerobic, and grows on the usual culture-media at room-temperature, and more rapidly at 37° C. It flourishes best in media containing sugar, and slowly liquefies gelatin. Milk becomes acid, the casein is firmly coagulated, and there is abundant separation of clear or slightly turbid serum and of gas-bubbles, with production of butyric acid. On potato there is also growth with gas-production. In stab sugar agar cultures the colonies are rather large, grayish-white, and oval or spherical, and they may present projecting threads or spicules. Many gas-bubbles are formed, and the medium is often split up into several fragments by the gas-formation. Bouillon is simply clouded. Of course, all these cultures must be grown in an atmosphere of hydrogen or in the Buchner anaerobic jars in order to obtain the foregoing results.

Fermentation Formula.—Glucose, lactose, and saccharose bouillon are all fermented by Bacillus aërogenes capsulatus. Mannite is not fermented. Glucose and lactose form about 40 per cent. of gas, and saccharose about 30 per cent., consisting of one part of CO_2 to two parts of H, with a slight amount of nitrogen. No foul odor occurs.

Pathogenesis.—The organism, according to Welch,[38] is quite virulent for guinea-pigs, pigeons, and sparrows, causing necrosis of tissues, bloody edema, and rapid development of gas. Rabbits are less susceptible, but may succumb to subcutaneous inoculation. Lanier produced gaseous phlegmons around the broken bones of rabbits by intravenous inoculations of pure cultures of the gas bacillus.

Welch first became acquainted with the gas bacillus in connection with the foamy organs ("Schaumorgane"), and the presence of gas in the blood in autopsies performed only a few hours after death without evidence of ordinary decomposition. This condition usually shows numerous gas-bubbles in the blood and viscera, especially the liver, with emphysema of the tissues.

Although in this condition the bacilli usually enter the circulation and viscera after death, yet there is evidence in favor of the view that they occasionally enter these tissues during life. This gas bacillus is also the cause of emphysematous gangrene, gaseous abscesses of the subcutaneous tissue, infections of the urinary tract, fibropurulent meningitis, and postmortem cavities in the brain. It has also entered the body from the gastro-intestinal tract, causing serofibrinous peritonitis with gas abscesses in the liver, and interstitial emphysema of the walls of the intestines and other canals. It has likewise been found in connection with "gas sepsis" or puerperal endometritis, even causing physometra, or distention of the uterine cavity with gas, and emphysema of the uterine walls.

The gas bacillus is the most widely distributed of bacteria, having been found in human and animal intestines and feces, in milk, earth, and other sources. It is a very widely distributed bacterium, and must be considered as an important pathogenic intestinal bacillus.

Bacteria which Resemble Bacillus Aërogenes Capsulatus.—"The bacillus of malignant edema can be distinguished from the gas bacillus by being thinner and by growing out into long filaments. It always produces spores, does not stain by Gram's method, is motile, liquefies gelatin more rapidly, produces a foul odor, generates less gas in lactose bouillon, clots, and then peptonizes casein, generates little or no gas in rabbits, and, by subcutaneous inoculation in susceptible animals, causes spreading bloody edema with little or no development of gas-bubbles" (Welch, *l. c.*). The bacillus of "Rauschbrand," which belongs to the same group, is not known to be pathogenic for man, but it closely resembles the gas bacillus.

Since the first description by Welch, in 1891–'92, of Bacillus aërogenes capsulatus, the same bacillus has been repeatedly encountered by other investigators, who, in ignorance of Welch's work, have assigned to it different names, and thereby created much confusion. E. Fränkel's "Bacillus phlegmones emphysematosæ," first described by Fränkel in 1893, is identical with Bacillus aërogenes capsulatus. The bacillus described by Veillon and Zuber in 1898, and subsequently by other French writers, under the name "Bacillus perfringens," without any reference to the American work, is likewise the same as Bacillus aërogenes capsulatus. The same is true of Lindenthal's "Bacillus emphysematis vaginæ," reported in 1897.

Klein, who likewise appears ignorant of the American work on Bacillus aërogenes capsulatus, in December, 1895, reported the isolation from the stools of several patients with diarrhea in St. Bartholomew's Hospital, London, of an organism to which he gave the name "Bacillus enteritidis sporogenes." He subsequently found the same organisms in cases of diarrhea in children, and cultivated it from milk and from dung. In pathogenic effects and in most of the morphological and cultural properties this bacillus, in what Klein calls "typical" cultures, resembles Bacillus aërogenes capsulatus, but it differs from it in the presence of motility. As regards this point, Klein says "the motility is little marked, and is seen only in a few rods, and is then not very lively." Under certain cultural conditions the bacillus forms spores abundantly, and under others not at all, and under certain conditions it peptonizes milk, which Welch's bacillus never does. In view of the identical pathogenic effects, the similar distribution, and the similar morphological and cultural characters, it is probable that Klein had in his cultures Bacillus aërogenes capsulatus, mingled perhaps with some contaminating motile bacterium and a peptonizing bacillus. Klein's various publications are in the "Centralbl. f. Bakteriol.," Bd. XVIII, XXII, and XXV, and in the "Local Government Reports," London, 1895–1900.

"Granulobacillus saccharobutyricus immobilis liquefaciens," described by A. Schattenfroh and R. Grassberger in the "Arch. f. Hyg.," 1900, Bd. XXXVII, page 54, as occurring in milk, water, earth, dung, etc., corresponds to the gas bacillus in all its characteristics, including lack of motility. They conclude[37] that it is a widely distributed saprophyte, and identify it with Fränkel's bacillus of gas phlegmon, which, as already stated, is itself identical with the bacillus previously described by Welch. Here, again, foreign authors fail to acknowledge Welch's priority, being apparently ignorant of his work and that of other American investigators on this organism.

THE PATHOGENIC PROTOZOA OF THE INTESTINES.

Amebæ were first found in the stools of cholera, and even in normal feces, but they were not identified with any special disease until Lösch[27] described them in connection with a certain form of dysentery in Russia in 1875. These organisms were later found in 500 cases by Kartulis,[28] and the cause of the disease, as well as its exact clinical and pathological lesions, have been most accurately described by Councilman and Lafleur.[29] This form of dysentery has been observed in America, Italy, Egypt, and Japan, and is occasionally accompanied by abscesses of the liver and lungs. The organisms can be seen in specimens taken from visceral abscesses, as well as from the undermined ulcer in the mucous and submucous coat of the intestines (see chapter on Dysentery).

Biological Properties.—The protozoan belongs to the subdivision of rhizopoda, and its diameter varies from 10 to 50 μ. The ameba at rest consists of a granular refractive sphere, but when motile, it shows two distinct portions or zones.

The central portion of the protoplasm is called the entoplasma, or endosarc, and it is coarsely granular and contains numerous vacuoles and foreign bodies, such as red blood-corpuscles, fat globules, granules of pigment, and bacteria. The outer zone is called the ectoplasma, or ectosarc, and is clear, refractive, homogeneous, and free from granules and other debris. The ameba has a nucleus which can be stained after the death of the organisms, or in tissues by means of eosin, acid fuchsin, or safranin, and often a nucleus can be seen in fresh specimens.

The Amœba coli constantly changes its shape, and throws out finger-like projections of protoplasm called pseudopodia. This causes the motility of the organism and its characteristic power of locomotion. The projection of the pseudopodia consists at first in a putting-out of the clear ectoplasma, but the granular entoplasma soon flows in and fills up the clear space.

A satisfactory demonstration of the pure cultivation of amœba coli has never been made, since the cultures were either not free from bacteria or the amebæ cultivated were derived from the normal intestine, or the hay, straw, fuscus, manure, or other material used as a culture-media. A very complete discussion of the cultivation of amebæ of various types will be found in the fourth section of the second volume of the 1896 edition of Flügge's "Die Mikroorganismen," and C. O. Miller, in "Contributions to the Science of Medicine by the pupils of William H. Welch," 1900, page 511, gives an account of the cultivation of amebæ.

Lesions Produced.—The lesions produced by Amœba coli are usually found in the large intestine, and the ulcers are not the superficial flat ulcers of ordinary dysentery, neither is a fibrinous diffuse inflammation present.

The intestinal ulcers of amebic dysentery are characteristic, and are described by Councilman and Lafleur as usually causing a nodular projection containing small cavities filled with a gelatinoid pus. This cavity opens upon the mucous membrane by means of a small orifice or sinuous tract, through which pus can be squeezed. This pus almost always contains amebæ in varying quantities. These ulcers are usually in the transverse or descending colon, but the lesion may also consist in an ordinary sloughing ulcer, or one with undermined edge or smooth edge and base.

The microscopic changes at the edges of the ulcer consist in dilation of the blood-vessels and degeneration of the glandular cells, together with infiltration with leukocytes. No fibrinous exudate is found on the surface in these cases. The submucous coat around the ulcer is also generally infiltrated with newly formed connective-tissue cells, and the central purulent cavity contains pus-cells, granular debris, bacteria of various kinds, and small round lymphoid cells and larger connective-tissue cells.

In the most usual form of ulcer the most characteristic change is the folding over and turning in of the mucous membrane, often almost covering the ulcer, and allowing only a small opening on the surface. Numerous nuclear figures are also present, showing active proliferation of the epithelial cells. It will thus be seen that the most intense changes take place in the submucous coat (see illustration of histology of amebic ulcer, Chap. xx, on Dysentery).

The amebæ are most numerous in the contents of the abscesses, but are also found in the mucous membrane, the muscular coat, and in the lymphatics and thrombosed blood-vessels. Councilman and Lafleur believe that the primary reaction to the ameba is the formation of granulation tissue, and that when real pus forms, it is secondary infection, due to the presence of pus-organisms.

The abscesses of the liver and lung, which almost always accompany this disease, are characterized by the absence of true pus consisting of leukocytes, and by a failure in the production of granulation tissue. The amebæ seem to exert a necrotic and solvent action on the tissues, causing remarkable destruction and breaking down of cells and tissue, with the formation of nuclear fragmentation and detritus. This causes the structureless, granular, broken-up material so characteristic of amebic abscess of the liver and lung. The amebæ are always present in these situations.

Reproduction in Animals.—A number of experiments in animals have shown that the introduction of amebæ into the intestines will cause conditions more or less resembling amebic dysentery. Lõsch, Kartulis, and others have produced a hemorrhagic inflammation of the large intestine by injecting amebic feces into the rectum of dogs. Kruse and Pasquale[30] injected from two to ten cubic centimeters of amebic feces into the rectum of sixteen cats, and obtained a number of positive results. They caused ulceration and hemorrhagic, mucopurulent exudation of the large intestines, and the animals usually died. The amebæ were constantly recovered from the bloody or mucopurulent discharges. In three instances they produced similar results by using the pus from liver abscesses, which contained only amebæ, and which was proved free from bacteria and other organisms by means of the microscope and cultures. These experiments with the pus from the liver were free from any error due to the admixture of other organisms, and show that the Amœba coli will produce a disease in animals similar to that of human beings.

Amebæ are at times found in the normal stool, and even in simple inflammations of the intestines, but these, when injected into animals, give negative results. They can not be distinguished from pathogenic amebæ in any other way, since their morphology is similar, and the pure cultivation of amebæ is as yet unsatisfactory. These harmless amebæ are known as Amœba coli mitis (harmless).

The Cercomonas intestinalis, Paramecium coli, Megastoma entericum, Trichomonas intestinalis, together with other rare protozoa have been described and pictured in chapter XXXIII on Intestinal Parasites, to which I refer for a more detailed account.

THE INFLUENCE WHICH BACTERIA EXERT ON THE PROCESSES OF DIGESTION.

The chyme, after it has passed from the stomach into the duodenum, is attacked by the secretion from the pancreas. This secretion contains three ferments, or enzymes, which act upon all the three primary foods (see chapter on Pancreatic Digestion).

The amylopsin acts upon the starches, converting them into maltose and dextrose, though absorption may take place at any stage of amylolysis after amylodextrin has been formed.

Trypsin, the second pancreatic enzyme, has an especial effect upon albuminous and albuminoid bodies, converting them finally into peptones after they have undergone a series of preliminary phases. But proteolysis is not arrested at the stage of peptone, which, if unabsorbed, undergoes further a breaking-down into leucin and tyrosin.

Steapsin is the fat-splitting ferment which changes fats, after they have taken up water, into glycerin and a free acid, such as palmitic or butyric acid. A large amount of fat is emulsified by the pancreatic juice and the bile, and is thus more easily absorbed.

The intestinal secretion, or succus entericus, is an alkaline liquid which has a fermentative effect upon the carbohydrates in the intestines, aiding the pancreatic secretion in converting the starches into sugar. This secretion also contains an inverting enzyme, which changes saccharose into dextrose and levulose, and maltose into dextrose. The disaccharids, such as cane-sugar and milk-sugar, both constituents of daily food, and maltose, the end-product of the digestion of starch, are thus turned into monosaccharids. These latter substances are readily soluble, and are thus easily absorbed and become available for nutrition.

The secretion of the large intestine mainly consists of mucus from the glands of Lieberkühn, and when the contents of the small intestine pass into the large intestine, the process of absorption and bacterial decomposition are the principal phenomena of importance.

It is difficult to determine with exactness whether bacteria play any part in the digestion, and yet certain bacteria are found at times in the intestinal contents which will produce effects in the test-tube similar to those of certain digestive ferments.

Bacillus subtilis has often been found in the intestine, and it is known that this organism will quickly liquefy solid gelatin or blood-serum in a test-tube. This is partially due to the fact that the albumin of the gelatin is reduced to a peptone by means of the ferment produced by bacillus. Bacillus liquefaciens ilei and Bacillus aërogenes capsulatus of the intestine will also peptonize gelatin. These and other bacilli may thus augment the effect of pepsin and trypsin in reducing proteids to the various peptones and their derivatives, such as leucin and tyrosin.

Streptococcus coli gracilis and Streptococcus coli brevis also liquefy gelatin, and these have both been found in the intestinal contents. All these organisms may be of some service in reducing albumin to peptones, but they also, together with other organisms, might perform a more important service to the economy.

This function would consist in the further reduction, by decomposition, of the tyrosin to a number of complex substances ending in phenol, while many other substances, such as indol, skatol, the ammonia salts of valerianic and butyric acid, and such gases as H, CO_2, H_2S, CH_4, and N would also be produced.

Certain special bacteria may cause the decomposition and putrefaction of albumin in the intestine, and one of the most important is Bacillus putrificus coli of Bienstock, which seems to be constantly present. This quickly attacks a solution of albumin in the test-tube, and forms ammonia, amido (fatty) acids, tyrosin, phenol, indol, and skatol. These processes will take place even when oxygen is excluded, a condition present in the intestines.

These bacteria of albuminous decomposition are possibly of service in reducing the excess of peptones or other albuminous derivatives to simple substances, some of which are not absorbed, but are simply cast off in the feces. The absorbent apparatus is thus relieved of an excess of absorbable material which might become burdensome.

Only a portion of the skatol and indol is cast off in the feces, the rest being absorbed into the blood, and eliminated later by the urine as skatoxyl-sulphuric acid and indican, the so-called ethereal sulphates. The exact value of these substances to the body is not known (see chapter on Intestinal Autointoxication).

As it is impossible to render the intestinal canal aseptic by experimental means (Hemmeter, "Études sur l'Action digestive de l'Intestin," Thirteenth International Congress of Medicine, Paris, August, 1900), we can not determine to what extent the character of animal digestion would be modified if carried out exclusively by the normal proteolytic, amylolytic, and adipolytic ferments of the intestinal canal (including the milk-curdling ferments in the gastric and pancreatic juices). Nuttall and Thierfelder (*l. c.*) have shown, it is true, that guinea-pigs can thrive on a perfectly sterile digestion, the ingoing food and the outgoing feces remaining sterile in their experiments. But the diet in these experiments consists of milk exclusively, and what the digestive results would be with a more varied diet—whether the digestive secretions could successfully transform a mixture of proteids, carbohydrates, and fats in the total absence of bacteria—is a question upon which the work of Nuttall and Thierfelder throws no light. These experiments were not conducted sufficiently long to prove conclusively that the animals on which they were carried out could exist indefinitely with an absolutely sterile intestinal canal. While we do not know what the digestive secretions accomplish without bacteria except from experiments in test-tubes,—and even in these there is no reliable evidence that bacteria have been excluded,—we have at least one observation which demonstrates the digestive action of the intestinal bacteria in the absence of proteolytic, amylolytic, and adipolytic ferments.

The Influence of the Intestinal Bacteria on the Digestion of Protein Substances in the Absence of Digestive Secretions.

The influence of bacteria on intestinal proteolysis has been investigated by Busch[39] at the surgical clinic of the University of Bonn.

The person upon whom the investigations were made had been gored by a mad steer in such a way that a fistula had been formed opening into the upper part of the jejunum, so that the entire gastric chyme, together with the bile, pancreatic juice, and the secretion of Brunner's glands, escaped exteriorly and none of these substances could enter the lower section of the small intestine. Although large quantities of food substances were taken by the mouth, this patient seemed doomed to death from inanition, because the food materials, after they had undergone gastric digestion and become mixed with the pancreatic juice and bile, escaped exteriorly before any further digestion or absorption could take place. Busch then resorted to feeding the patient by stuffing liquid and semisolid food into the lower section of the opened intestinal canal. Curiously enough, he succeeded in restoring the strength and weight of the patient, and in maintaining him in the nitrogenous equilibrium. This rapid recuperation by nourishment being introduced into the lower opening of the fistula proves that digestion can be carried on in the intestinal canal in the entire absence of gastric or pancreatic juice, bile, or the secretion of Brunner's glands. The digestive process could have been effected either by the succus entericus, which, according to Demant ("Virchow's Archiv," No. 75), possesses no proteolytic properties, or by micro-organisms. Busch seemed inclined to attribute to the succus entericus the power of dissolving coagulated egg-albumen, but in none of his experiments can the action of the proteolytic micro-organisms be eliminated. Concerning the action of the succus entericus on protein substances, most investigators agree that it is without influence on boiled white of egg and meat; but according to Thiry ("Wien. Sitzungsber.,"[80] Bd. L), it has the power of dissolving fibrin. It can not transform albumoses into peptone (Wenz, "Zeitschr. f. Biol.," Bd. XXII). The literature given in this latter article, and also that in Hammarsten ("Lehrbuch d. physiol. Chem.," 1899, S. 285), of those investigators who attribute a proteolytic action to the succus entericus has been gone over critically by Hemmeter. But in none of them can the action of bacteria be eliminated. This is particularly true of the experiments of Schiff ("Centralbl. f. d. med. Wissensch.," 1868, S. 367) and those of Gachet and Pachon ("Arch. de physiol.," Bd. V, S. 10). When the succus entericus was collected from animals and sterilized by filtration through a Pasteur filter and saturated with crystals of thymol, and the experiments were conducted under aseptic precautions, the succus entericus exhibited no proteolytic action. Hemmeter is, therefore, disposed to attribute any proteolytic action recorded in the experiments of Busch to the action of proteolytic micro-organisms. Busch inclosed coagulated egg-albumen in small gauze sacs, and determined both the wet and the dry weight of the precipitated albumin used in his experiments. Attaching a string to the sacs, he permitted them to descend into the lower opening of the fistula. After they had remained in the intestine for about five hours he withdrew them by means of the string, and determined the weight of the dried residue of albumin that had remained, and found that a loss of weight had taken place varying from 5 to 35 per cent. of the introduced substance. Brücke (*l. c.*) also favors the view that this proteolytic digestion is due to the intestinal bacteria.

The sugars in the intestines might also be acted upon by bacteria or yeasts, and this takes place principally in the small intestine. The products of this fermentation are acetic acid, butyric acid, lactic acid, alcohol, hydrogen, and carbon dioxid.

Bacillus coli communis, the constant and characteristic intestinal bacterium, will ferment glucose, forming hydrogen, carbon dioxid, and ethyl alcohol, and this germ also ferments lactose and saccharose, likewise Bacillus lactis aërogenes. The colon bacillus also produces acetic and lactic acid, the latter from lactose as well as dextrose.

Bacillus butyricus, an inhabitant of the intestine, ferments starch, dextrin, and various sugars, forming butyric acid, carbon dioxid, and hydrogen, and it can even ferment cellulose. It will thus be seen how the sugars could in part be reduced to a simple substance, such as alcohol, or transformed into acids and gases, which are cast off in the feces.

Not much is known of the effect which the bacteria of the intestines have upon the fats, but they may aid in reducing them to glycerin and the fatty acids.

From what has gone before it will be seen that while some of the intestinal bacteria could aid in the digestion of peptones, yet their more important rôle would seem to be of a different nature. It would seem probable that they come to the aid of the chemical digestive ferments and reduce the excess of ingested food to simple compounds, which are either absorbed into the blood or cast off as worthless in the feces.

Cushing and Livingood[5] have recently published some very interesting observations which they have made upon the bacteria which are present in the various portions of the small intestine of men and animals, and they have demonstrated that the variety and number of bacteria in the various portions of the canal vary greatly. They first point out that Nuttall and Thierfelder[32] have shown that a guinea-pig which was removed by cesarean section, kept in a sterile chamber, and fed on sterile food was kept alive for eight days. At the end of this time it was killed, and its intestines were found to be free from bacteria. Levin[33] also showed that in the arctic regions, where bacteria are very rare, the intestines of such animals as white bears, seals, reindeer, etc., are almost always sterile. They believe that the variety of organisms in the intestines is greatly influenced by the food, and that the colon bacillus and one or two other varieties are the only constant inhabitants of the intestinal canal. The other varieties are inconstant and are usually introduced in the food, and during early life it would even seem that assimilation of food can take place without the presence of bacteria.

It is certainly true, as they state, that the organisms in the feces do not represent the bacterial flora in those portions of the intestines where active digestion takes place. It should also be borne in mind that many of the bacteria which can be stained in cover-slip preparations can not be cultivated.

In order to clear up these doubtful points, Cushing and Livingood made a study of the bacteria in the various portions of the small intestine of dogs, and they found that the relative number of bacteria at different situations and periods of digestion and rest remained quite constant. In one dog, after a twenty-four-hour fast, they found the stomach sterile by culture and 8 colonies in the duodenum. The jejunum, 50 cm. below the stomach, contained 3 colonies; 100 cm. below it gave 174; and 157 cm. below it was sterile. The ileum, 247 cm. below the stomach, contained 63 colonies; 337 cm. below it showed 825 colonies; and 10 cm. above the ileocecal valve it contained 5000 bacteria. The cecum contained 100,000 colonies, and the rectum showed 750 colonies.

This shows that the upper part of the intestine is much freer from bacteria than the lower portion of the canal, and that the upper portion can be made almost amicrobic by fasting, thus emptying the intestines of the products of digestion. They also made autopsies on or cultures from a number of dogs, and found the bacterial flora to be very limited. They concluded that the intestinal flora is restricted to a few constant forms, and this work would seem to prohibit the acceptance of the theory that the intestinal bacteria exert any essential effect on digestion and nutrition, as most of the bacteria which have been described by other authors were not found as constant intestinal dwellers. After all, the subject is still an obscure one, and until media are devised which will cultivate most of the intestinal species, we shall still remain in doubt as to just what rôle the various bacteria play in the processes of digestion and assimilation. Many careful experiments must also demonstrate just what products each bacterium causes, both in the intestine and in the test-tube. At present our exact knowledge is scanty, but the subject offers a most tempting field to physiologists, chemists, and bacteriologists.

LITERATURE.

1. "Coccobacteria septica," p. 94.
2. "Zeitschr. f. klin. Med.," Bd. III, 1881.
3. "Arch. f. Hyg.," Bd. IV, 1886.
4. "Die Mikroorgan. der Mundhöhle," Leipzig, Thieme, 1892.
5. "Contributions to the Science of Medicine," dedicated to Wm. H. Welch, 1900.
6. "Johns Hopkins Hospital Reports," vol. VI, 1896.
7. "Fortschr. d. Med.," Bd. LXXXV, und "Darmbact. des Säuglings," Stuttgart, 1886.
8. "The Fermentation Tube," Wilder, "Quarter Century Book," 1893; "Amer. Jour. of the Med. Sci.," September, 1895.
9. "Semaine Med.," October 20, 1897.
10. "Zeitschr. f. klin. Med.," Bd. VIII, 1884.
11. "Zeitschr. f. Hyg.," Bd. XXX, H. 2, 1899.
12. "Centralbl. f. Bakteriol.," Bd. XXIV, No. 22–24, 1898.
13. "Bulletin of the Johns Hopkins Hospital," vol. XI, No. 115, October, 1900.
14. "The Serum Diagnosis of Disease," Wm. Wood & Co., N. Y.
15. Hill, "Public Water-supplies," 1898.
16. "Centralbl. f. Bakteriol.," I. Abth., Bd. XXVI, No. 18, 19.
17. "Zeitschr. f. Hyg.," Bd. XXXI, 1899.
18. "Jour. Amer. Med. Assoc.," June 10, 1899.
19. "Jour. of Exper. Med.," vol. III, No. 6, 1898.
20. "Jour. of Exper. Med.," November, 1897.

21. "Verhältniss. mikros. Organismen z. Cholera," "Zeitschr. f. Biol.," Bd. VIII.
22. "Zeitschr. f. Hyg.," Bd. XII, H. 1, 1895.
23. "Berlin. klin. Wochenschr.," H. 7, 1899.
24. "Jour. of Exper. Med.," vol. III, 1898.
25. "Arch. f. exper. Path.," Bd. XXXII.
26. "Zeitschr. f. Hyg. und Infek.," Bd. XVIII.
27. "Virchow's Archiv," Bd. LXV, 1875.
28. "Virchow's Archiv," Bd. CV, 1885.
29. "Johns Hopkins Hospital Reports," 1891.
30. "Zeitschr. f. Hyg. und Infek.," No. 15, 16.
31. "Fortschr. d. Med.," Bd. III, 1885.
32. "Zeitschr. f. physiol. Chem.," Bd. XXI, 1896.
33. "Annales de l'Institut Pasteur," tome XIII, 1899.
34. "Bulletin of the Johns Hopkins Hospital," July, 1900.
35. "Phila. Med. Jour.," September 17, 1898.
36. "Bulletin of the Johns Hopkins Hospital," 1892, vol. III, p. 81.
37. "Münch. med. Wochenschr.," July 31, 1900.
38. "Bulletin of the Johns Hopkins Hospital," September, 1900.
39. "Vorlesung über Physiologie," E. Brücke, S. 351.

40. Abel, Rudolf, "Ueber das Vorkommen feiner Spirillen in Dejectionen," "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. XV, 1894, No. 7.

41. Aufrecht, "Ueber den Befund feiner Spirillen," u.s.w., "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. XV, 1894, No. 12.

42. Arnaud, O., "Recherches sur l'étiologie de la dysenterie aiguë des pays chauds," "Annales de l'Institut Pasteur," Bd. VIII, 1894, No. 7.

43. Babes, V., "Ueber die durch Streptokokken bedingte gelbe Leberatrophie," "Virchow's Archiv," Bd. CXXXVI, 1894.

44. Blachstein, A. G., "Intravenous Inoculation of Rabbits with Bacillus coli," etc., "Bulletin of the Johns Hopkins Hospital," Baltimore, vol. II, 1891, No. 14; ref. im "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. XII, S. 278.

45. Bernheim, A., "Ueber den Befund des Bakterium coli in einem Panaritium bei Typhus abdominalis," "Centralbl. f. klin. Med.," Bd. XIV, 1893, S. 13.

46. Bunge, R., "Zur Kenntniss der geisseltragenden Bakterien," "Fortschr. d. Med.," Bd. XII, 1894, No. 17.

47. Brunner, Conrad, "Eine Beobachtung von Wundinfektion durch das Bakterium coli communis," "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. XVI, 1894, No. 24.

48. Breslau, "Ueber Entstehung und Bedeutung der Darmgase bei neugeborenen Kindern," "Monatsschr. f. Geburtsk. u. Frauen-Krankheiten," Bd. XXVIII, 1866.

49. Boas, J., "Diagnostik u. Therapie der Magenkrankheiten," 1. Theil, 3. Aufl., 1894.

50. Booker, William, "A Study of Some of the Bacteria Found in the Dejecta of Infants," etc., "Transactions of the Ninth International Med. Congress," vol. III; ref. im "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. V, 1889, S. 316.

51. Baumgarten, "Ueber die Uebertragbarkeit der Tuberculose durch die Nahrung," "Centralbl. f. klin. Med.," 1884, No. 2.

52. De Bary, W., "Beitrag zur Kenntniss der niederen Organismen im Mageninhalte," "Arch. f. exper. Path.," Bd. XX, 1886, S. 243.

53. Bienstock, B., "Ueber die Bakterien d. Fäces," "Zeitschr. f. klin. Med.," Bd. VIII, 1884.

54. Billroth, Th., "Coccobacteria septica," p. 94.

55. Brieger, L., "Ueber Spaltungsproducte der Bakterien," "Zeitschr. f. physiol. Chem ," Bd. VIII, 1883–'84.

56. Brieger, L., *ibid.*, Bd. IX, 1885.

57. Buchner, Hans, "Beiträge zur Kenntniss der Neapeler Cholerabacillen," Arch. f. Hyg.," Bd. III, 1885.

58. Ciechomski, A., u. Jakowski, M., "Ungewöhnlich lange dauernder künstl. After," u.s.w., "Arch. f. klin. Chirurgie," Bd. XXVIII, 1894, 1. Heft.

59. Carbone, Tito, "Sui veleni prodotti del Proteo volgare," "Rif. med.," Settembre, 1890; ref. in "Baumgarten's Jahresb.," Bd. VI, 1890.

60. Cadéac, C., et Bournay, J., "Rôle microbicide des sues digestifs," etc., "La province medicale," Bd. VIII, 1893, No. 28; ref. im "Centralbl. f. Bakteriol. u. Parasitenk.," 1894, 15, 16.

61. Capitan et Morau, "Recherches sur les microorganismes de l'estomac," "Compt rend. Soc. de biol.," 1889, p. 25.

62. Demel, C., u. Orlandi, "Die Serumtherapie und das Bakterium coli communis," "Centralbl. f. Bakteriol. u. Parasitenk.," 1894, No. 6.

63. Denys, J , et Brion, "Etude sur le principe toxique du Bact. lactis aërogenes," "La Cellule," Bd. VIII, 1892; ref. im "Centralbl. f. Bakteriol. u. Parasitenk ," Bd XVI, No. 3.

64. Denys, J., et Brion. E., "Sur les rapports du Pneumobacille de Friedländer," etc , "La Cellule," Bd. IX, 1893; ref., *ibid.*

65. Dmochowsky u. Janowski, "Zwei Fälle von eiteriger Entzündung der Gallengänge," u.s.w., "Centralbl. f. pathol. Anat. u. exper. Path.," 1894, No. 4.

66. Dreyfus, R., "Ueber die Schwankungen in der Virulenz des Bacterium coli," Inaug. Diss., Strassburg, bei Gebweiler, 1894; cit. nach "Centralbl. f. Bakteriol. u. Parasitenk.," 1894, No. 14.

67. Escherich, Th., *ibid.*, No. 43 ("Ueber Vibrio felinus").

68. Emmerich, Rudolf, "Untersuchungen über die Pilze der Cholera Asiatica," "Arch. f. Hyg.," Bd. III, 1885.

69. Escherich, Th., "Aerztl. Intelligenzblatt," 1884, No. 54.

70. Escherich, Th., "Ueber das Vorkommen von Vibrionen im Darmkanal," u.s.w., *ibid.*, No. 45.

71. Escherich, Th., "Beiträge zur Kenntniss der Darmbakterien," "Münch. med. Wochenschr.," 1886, No. 1.

72. Escherich, Th., "Die Darmbakterien des Neugeborenen und Säuglings," "Fortschr. d. Med.," Bd. III, 1885, No. 16 u. 17.

73. Eisenhart, H., "Puerperale Infektion," u.s.w., "Arch. f. Gynäkologie," Bd. XLVII, 1894.

74. Gärtner, "Ueber die Fleischvergiftung in Frankenhausen," u.s.w., "Correspondenzbl. d. allg. ärztl. Vereins von Thüringen," 1888, No. 9; ref. in "Baumgarten's Jahresb.," Bd. IV, 1888.

75. Gessner, C., "Ueber die Bakterien im Duodenum des Menschen," "Arch. f. Hyg.," Bd. IX.

76. Gruber. "Eine Methode der Kultur anaërobischer Bakterien," u.s.w., "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. H, 1887, No. 12.

77. Hodenpyl, A., "On the Etiology of Appendicitis," "New York Medical Journal," vol. LVIII, 1893, p. 777; ref. im "Centralbl. f. Bakteriol. u. Parasitenk.," 1894, No. 21.

78. Henke, F., "Beitrag zur Verbreitung des Bacterium coli commune in der Aussenwelt," "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. xvi, 1894, No. 12.

79. Jeffries, John A., "Ein Beitrag zu dem Studium der Sommerdiarrhoen bei Kindern," "Arch. of Pediatrics," 1889–'90; ref. im "Arch. f. Kinderheilk.," Bd. xiii, 1891.

80. Jeanselme, "Contribution à l'étude des thyroidites infectieuses," "Arch. gén. de med.," 1893; ref. im "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. xvi, 1894, No. 21.

81. Jaeger, H., "Die Aetiologie des infektiösen fieberhaften Icterus," "Zeitschr. f. Hyg.," Bd. xii, 1892.

82. Kemper, W., "Ueber den vermeintlichen Antagonismus," u.s.w., "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. xvii, 1895, No. 1.

83. Kuhn, F., "Morphologische Beiträge zur Leichenfäulniss," "Arch. f. Hyg.," Bd. xiii.

84. Krogins, A., "Recherches bacteriologiques sur l'infection urinaire," Helsingfors, 1892; ref. im "Baumgarten's Jahresb.," Bd. viii, 1892.

85. Kuisl, "Beiträge zur Kenntniss der Bakterien im normalen Darmtractus," Inaug. Diss., München, 1885.

86. Lubarsch, O., "Ein Fall von sept. Pneumonie," u.s.w., "Virchow's Archiv," Bd. cxxiii, S. 70.

87. Lesage et Macaigne, "Choléra en 1892," "Annales de l'Institut Pasteur," Bd. vii, 1893.

88. Lermoyez, "Un cas d'amygdalite coli bacillaire," "La Semaine méd.," 1894, No. 37.

89. Lesage, A., "Du bacille de la diarrhée verte des enfants du premier age," "Arch. de physiologie," 1888, No. 1.

90. Messea, "Contribuzione allo studio delle ciglie," etc., "Rivista d'Igiene e sanita pubblica," 1, No. 14; ref. in "Baumgarten's Jahresb.," Bd. vii, 1891.

91. Miller, W. D., "Die Mikroorganismen der Mundhöhle," 2. Aufl., Leipzig, bei Thieme, 1892.

92. Minkowski, D., "Ueber die Gährungen im Magen," "Mittheilungen aus der med. Klinik zu Königsberg," 1. Pr.; herausgegeben von B. Naunyn, Leipzig, bei Vogel, 1885.

93. Macfadyen, Nencki, u. Sieber, "Untersuchungen über die chemischen Vorgänge im menschlichen Dünndarm," "Arch. f. exper. Path.," Bd. xxviii, 1891.

94. Maggiora, A., "Einige mikrosk. u. bakt. Betrachtungen," u.s.w., "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. xi, 1892.

95. Marpmann, "Zur Unterscheidung des Bakt. typhi abd. vom Bakt. coli comm.," "Centralbl. f. Bakteriol. u. Parasitenk.," Bd. xvi, 1894, No. 20.

96. Metschnikoff, "Ueber Immunität gegen Cholera," "Wien. med. Presse," Bd. xxxv, 1894, No. 39.

97. Nothnagel, "Erkrankungen des Darmes und des Peritoneum."

98. Nothnagel, H., "Die normal in den menschlichen Darmentleerungen vorkommenden niedersten (pflanzlichen) Organismen," "Zeitschr. f. klin. Med.," Bd. iii, 1881.

99. Nicolle et Morax, "Technique de la coloration des cils," "Annales de l'Institut Pasteur." Bd. vii, 1893.

100. Nencki, "Ueber die Zersetzung der Gelatin und des Eiweisses," u.s.w., Bern bei Dalp, 1876.

101. Nocard, "Société de la biologie," Paris, "Progrès méd.," 1895.

102. Pisenti, G., u. Bianchi-Mariotti, G. B., "Beziehungen zwischen dem Bakterium coli commune u. der Typhusinfektion," "Centralbl. f. Bakteriol. u. Parasitenk.," 1894, No. 17.

103. Péré, "Bact. coli comm. et Bacille typhique," "Annales de l'Institut Pasteur," Bd. VI, 1892.

104. Pfeiffer, R., "Ueber die specifische Immunitätsreaktion der Typhusbacillen," "Deutsch. med. Wochenschr.," 1894, No. 48.

105. Posner u. Lewin, "Ueber Selbstinfektion vom Darm," aus "Berlin. klin. Wochenschr.," 1895, No. 6.

106. Van Puteren, "Ueber die Mikroorganismen in Magen von Säuglingen," Wratsch, 1888, No. 22; ref. im "Baumgarten's Jahresb.," 1888.

107. Schmidt, Alex., "Zur Kenntniss der Bakterien in den Säuglingsstühlen," "Wien. klin. Wochenschr.," 1892.

108. Salus, "Ein Fall von Grünfärbung des Stuhles durch den Bakterium pyocyaneus," "Prager med. Wochenschr.," 1894, No. 33.

109. Sanarelli, "Étude sur la fièvre typhoide expérimentale," "Annales de l'Institut Pasteur," Bd. VII, 1894, p. 353.

110. Schild, W., "Das Auftreten von Bakterien im Darminhalte Neugeborener," u.s.w., "Zeitschr. f. Hyg. u. Infektionskrankheiten," Bd. XIX, 1895.

111. Schnitzler, Jul., "Zur Aetiologie der Cystitis," Wien, bei Braumüller, 1892.

112. Sardoillet, Leouis, "Péritonite sans perforation et Bact. coli comm.," Paris, 1893; ref. im "Centralbl. f. Bakteriol. u. Parasitenk.," 1894.

113. Stahl, "Mikroorganismen in den Darmentleerungen," "Verhandl. des III. Cong. f. innere Med.," 1884.

114. Stern, R., "Ueber Disinfektion des Darmkanales," "Zeitschr. f. Hyg. u. Infektionskrankheiten," Bd. XII, 1892.

115. Sucksdorf, W., "Das quantitative Vorkommen von Spaltpilzen im menschlichen Darmkanal," "Arch. f. Hyg.," Bd. IV, 1886.

116. Stoecklin, "Recherches sur la mobilité et les cils," etc., "Annales Suisses de sciences médicales"; ref. im "Centralbl. f. Bakteriol. u. Parasitenk.," 1894, No. 3.

117. Terni u. Pellegrini, "Bakteriologische Untersuchungen ü. die Choleraepidemie in Livorno," u.s.w., "Zeitschr. f. Hyg. u. Infektionskrankheiten," Bd. XVIII, 1.

118. Utpadel, "Ueber einen pathogenen Bacillus aus Zwischendeckenfüllung," "Arch. f. Hyg.," Bd. VI, 1887.

119. Wesener, F., "Kritische u. exper. Beiträge zur Lehre von der Fütterungstuberculose," "Freiburger akad. Habilitätionsschrift," 1886, cit. nach "Baumgarten's Jahresb.," 1885.

120. Woodward, "The Medical and Surgical Report of the War of the Rebellion," vol. I, part II, 1879.

CHAPTER VIII.

METHODS AND TECHNICS OF DIAGNOSIS.

METHODS OF EXAMINATION.

Inspection.

Important information can be gained by systematic inspection of the abdomen, which should properly be made first with the patient in the erect and then in the horizontal position, and with good daylight. In the erect position one can observe not only partial bulgings, such as are caused by relaxation of the abdominal recti muscles, and those caused by the inflation of single intestinal loops, but uniform enlargement of the abdomen also appears more prominently when the patient is seated or standing than when he is lying on his back.

The enlargements, which often disappear entirely or nearly entirely in the recumbent position, and also during sleep and chloroform narcosis, are those occasionally observed, both in adults and in children, as a result of lordosis of the lumbar segment of the spine combined with insufficient development or with acquired debility of the abdominal recti muscles and spasm of the diaphragm ; according to Talma, this is not a very rare occurrence in hysterical persons.

During the examination of the condition of the abdominal skin, attention must be devoted to the occurrence of whitish striæ and of visible abdominal veins (the superior and the inferior epigastric veins), which are seen through the skin and are of a blue color, particularly when they are much distended. The striæ resemble scars, being shorter or longer streaks, usually parallel, which are caused by breaks in the continuity of the subcutaneous cellular tissue and the cutis, with consequent slight formation of cicatricial tissue. The more recent striæ have a reddish appearance, but the older ones are snow white, or may be slate color, as a result of the depositing of pigment. They are the results of a great distention of the abdominal walls, such as occurs, for instance, in frequent pregnancies, or as a sequence to great increases of the intra-abdominal pressure, in ascites, in quickly growing tumors of the abdominal organs, and also as a result of a great increase in the fat of the subcutaneous cellular tissue of the abdomen. The striæ are, therefore, an evidence of overstretching of the abdominal integument, and persist after the causes of the overdistention have been removed—for instance, after delivery, the extirpation of abdominal tumors, or the withdrawal of an ascitic accumulation.

Very much distended abdominal veins are observed only when the flow of the venous blood out of the lower extremities is rendered more difficult by an increase of the intra-abdominal pressure (ascites, quickly increasing tumors of the abdominal organs), by thrombosis or by compression of an iliac vein or inferior vena cava, or when advanced obstruction has occurred in the portal venous system—cirrhosis and other diseases of the liver, pylethrombosis, compression of the portal vein. When the obstruction is caused by thrombosis, the abdominal veins and the anastomosing thoracic veins are much distended, because a portion of the blood which otherwise flows through the inferior vena cava now takes its way through the epigastric veins, and through these into the superior vena cava, and thus reaches the right heart in a round-about way. In the case of advanced obstruction of the portal system, a corona of varicose distended veins is formed, with its base directed toward the navel, which is designated the "caput medusæ" (an extensive dilation of the superficial veins of the abdomen about the umbilicus, seen in cases of portal obstruction), or "cirsomphalos" (a varicose condition of the navel). According to Rokitansky, the origin of this condition is traced to the union of the portal vein region with the veins of the abdominal walls by means of the umbilical vein, which has again become permeable; or by means of the newly formed accessory portal vein branches.

Changes in the Form of the Abdomen.

In athletes, soldiers, etc., the abdomen is often seen slightly retracted, but under normal conditions (the time of pregnancy being, of course, excluded) the abdomen is in the same plane as the thorax, and does not project beyond it. In small, healthy children this does occur, however, because of the comparatively small development of the thorax. In scrofulous and rachitic children the large abdomen often appears in extreme contrast to the size of the thorax. In advanced old age, however, the abdomen is occasionally found retracted and concave, like a trough, as a result of emaciation and of senile atrophy of the abdominal organs, so that the ribs and the crest of the ilium project beyond it. The same thing is observed after prolonged and continued inanition. After the taking up of large amounts of liquid and solid foods, the volume of the abdomen increases; after thorough evacuations and the passage of flatus, it decreases.

Abnormal Enlargement of the Abdomen.—This may occur (*a*) uniformly or (*b*) in circumscribed areas.

Uniform Enlargement of the Abdomen.—The abdomen may be enlarged uniformly when large amounts of fat are collected in the abdominal walls and around the abdominal organs. Inspection alone will not enable us to decide whether meteorism, ascites, or great massing of fat is the cause of the abdominal enlargement; palpation, percussion, and even exploratory puncture are often necessary for this purpose. If the large abdomen is in striking contrast to the emaciated upper part of the body and to the thin extremities, we may exclude the great massing of fat in the abdominal walls as the cause of the enlargement, because the signs of obesity at the other parts of the body should be present as well. Uniform enlargement is most frequently observed when air or gases are collected in the intestines (meteorism); less frequently it is noticed when air or gas escapes out of the stomach or intestine into the peritoneal cavity in perforation and in meteorism of the peritoneum. In the case of intestinal meteorism, the abdomen as a whole is uniformly distended and the abdominal wall very much stretched at all points if a large number of the intestinal loops are excessively distended by gases. The causes of intestinal meteorism are enumerated in a special chapter. In most cases an atony of the intestinal muscles is at the base of the trouble—such as often occurs in typhoid fever, peritonitis, and advanced inflammation of the intestines. Other causes which may be mentioned are strictures of a cancerous, syphilitic, or tuberculous nature in the lower section of the intestines, invagination and axis rotation, coprostasis, fecal accumulation and coproliths, and tumors of neighboring organs which compress the intestines—all of which conditions will be described in separate chapters. Meteorism is favored by lordosis of the lumbar segment of the spine, combined with insufficient development or acquired weakening of the abdominal recti muscles and also with spasm of the diaphragm. Stricture in the lower part of the intestines is said to cause a uniform enlargement of the abdomen only when it has reached a very advanced stage. Before this occurs, only a very small part of the intestines—namely, that directly above the stricture—is distended by the solid and liquid ingesta and gases, and the abdominal wall at that point is partially distended. When gas passes out into the free peritoneal cavity as a result of perforation of the stomach or intestines, the increase in the volume of the abdomen is at first quite uniform; but after an abundant transudation of liquid into the peritoneum (ascites), the enlargement of the abdomen is not uniform, but varies according to the position of the body, so long as the distention of the abdominal walls has not reached its highest degree. Since the mobile fluid occupies the lowest parts of the abdomen by reason of its weight, in whatever position the patient may be the lowest points are always distended the most, so long as such a protuberance is yet possible. Undulatory wave motions which are communicated to the abdominal walls may also be detected on palpation. Different signs and appearances prevail when the tension of the abdominal walls has reached its highest stage: the abdomen then appears distended uniformly, like a sphere, and no change in form is noticed when the posture is changed.

Irregular Enlargement of the Abdomen.—A circumscribed distention of the abdominal wall may be caused by a larger inflation of the colon or of single parts of the same by typhlitic abscesses, tubercular glands of omentum or peritoneum, by large malignant neoplasms, and by fecal tumors. This partial enlargement can be more readily noticed in emaciated adult patients with thin, yielding abdominal walls, and in children. We are, of course, not justified, however, in excluding these diseased conditions if such a distention of the abdominal wall can not be demonstrated, because they may be overlooked under conditions previously mentioned, and the enlarging masses may be concealed under the ribs. It has also been stated that stricture in the lower part of the intestines occasionally at first causes only partial distention of the abdominal wall. In the case of umbilical hernia, a more or less extended spherical distention of the abdominal walls in the middle line of the body is noticed; in the so-called relaxation of the recti a band-like or sausage-formed partial enlargement is noticed at the same place. If, as a result of long-continued increase in the intra-abdominal pressure,—tumors, ascites, pregnancies,—the recti muscles have separated, the cleft between them offers a place of less resistance, into which intestinal loops force themselves transiently on a sudden change from the recumbent position to a standing or sitting attitude or in the case of a cough or straining. But in ascites and abdominal tumors intestinal loops may permanently force this cleft apart, and thus exceedingly distend the abdominal walls. That the distention is practically caused by parts of the intestines is proved by their tympanitic sound and the frequent recurrence of quick peristaltic movements at the place in question. This evidence is of importance in the diagnosis of large median herniæ. In a case referred to me by Dr. Charles E. Sadtler, the medium cleft between the abdominal recti was so wide as to permit the entire stomach to slip through when it was distended by air or CO_2. The woman had gone through several severe and protracted labors. Her stomach was in a position of extreme gastroptosis.

Diminution in the Size of the Abdomen.—A uniform diminution in the size of the abdomen, in which the same appears sunk in, like a trough, bounded by the sharply projecting ribs and the crest of the ilium, is observed in extreme emaciation resulting from exhausting sickness, after long-continued inanition, as well as in the case of stricture of the pharynx, esophagus, and the cardia; in the latter case it is observed as soon as no more food arrives in the stomach and intestines. The abdomen is retracted in a concave manner when extensive herniæ, including a large section of the intestines, have occurred, as well as in tetanic contractions of the intestinal muscles as a result of lead colic or basilar meningitis.

Partial depressions of the abdominal walls are sometimes noticed in spastic contractions of single intestinal loops. In advanced stricture of the pylorus and of the duodenum the upper part of the abdomen is, as a rule, much distended, the lower part, on the contrary, being drawn in, because both the large and the small intestine contract more and more when the chyme no longer reaches them.

Peristaltic Movements of the Intestines.—Under normal conditions the peristaltic movements of the stomach and intestines are not visible through the abdominal walls. If they are visible, we may assume an abnormal increase in the peristalsis of these organs. In intestinal colic, at the onset of lead colic, and when any occlusion has developed, there is increased peristalsis of the intestines. If occlusion or stenosis has existed for a long time, paralysis of the musculature results, and all visible peristaltic movements cease. Peristaltic unrest of the stomach may extend to the upper part of the intestines.

Electrodiaphany.—Transillumination of the Intestine.

In 1891 I gave a demonstration before the Clinical Society of Maryland, showing the possibility of outlining the colon by the electrodiaphane in a similar manner in which the stomach can be outlined by this method (see Electrodiaphany, "Diseases of the Stomach," second edition, by the author, p. 104). In 1896 I described a method by which the duodenum can be intubated in the human being ("Johns Hopkins Hospital Bulletin," vol. VII, No. 61, p. 79, 1896); this method and that of Dr. F. Kuhn and F. B. Turck are described in this work in the chapter on Duodenal Intubation. By means of the apparatus there described, the electrodiaphane can be introduced into the duodenum, and the location of this portion of the bowel can, as a rule, be determined in favorable cases, the conditions for favorable observations of this character being in a general way about the same as those which control gastrodiaphany.

For introducing the electrodiaphane into the duodenum I prefer the Kuhn metallic spiral sounds, which are very elastic, and with a little practice can be introduced into the duodenum if there is no pyloric stenosis (Franz Kuhn, "Sondierungen am Magen, Pylorus, u. Dünndarm des Menschen," "Arch. f. Verdauungs-Krankheiten," Bd. III, S. 19). Dr. Kuhn, who instructed me personally in the execution of this method during a visit to Baltimore in 1897, employs it also for the exploration of the colon.

Intubation and Sounding of the Rectum, Sigmoid, and Colon.—Properly constructed sounds can be passed through the entire colon and beyond the ileocecal valve, whereby the permeability of the large intestine can be established, because the Turck or Kuhn sounds can not kink upon themselves, and the mere possibility of introducing them for a certain distance means that the passage through the bowel must be unobstructed for that distance. Long before the publication of the method of sounding the colon by closely wound metallic spiral sounds sheathed in rubber tubes, similar to the ordinary tubes for gastric lavage, I employed the Langdon tube for this purpose. This tube contains no metallic spiral, but is simply composed of rubber, but having such thick walls that kinking is impossible even if one makes special efforts to effect this by forcible means outside of the body. At the same time the Langdon tube is no more likely to injure the colon than an ordinary lavage tube is likely to injure the stomach. The Langdon tube is less complicated and easier to disinfect. During the introduction it is essential that lukewarm water should be allowed to run through the tube into the colon, which greatly aids in pushing away folds of mucosa, fecal masses, etc., that would otherwise obstruct the onward course of the bowel end of the tube. The knee-chest position is preferable at the beginning of the introduction, but when, from the length of the tube, one may about judge that it has reached the splenic flexure, the patient should be made to rest on the right side—this position relieves the colon of any possible pressure from the liver. The onward pushing should be done cautiously and slowly and with constant slow current of water running through the tube. In very sensitive patients it is sometimes necessary to anesthetize the sphincter and lower rectum with a 3 per cent. solution of cocain, but I have, as a rule, been able to get along without this. The tubes containing metallic spirals possess one advantage in that they can be recognized by the X-rays and fluoroscope, and thus the entire location of the colon made out by the eye.

This demonstration requires extensive apparatus, however, and is not available for the general practitioner, in whose hands the Langdon tube will be more practical and less likely to injure the mucosa. When it becomes necessary to use the electrodiaphane in the colon, the same instrument can be employed as is recommended for illuminating the stomach, but it must be much longer, corresponding to the length of the colon. Herying and Reichmann ("Therapeut. Monatsh.," March, 1892) published the first investigations for determining the limits of the colon by means of diaphanoscopy. I have followed their advice in introducing about 1500 c.c. of lukewarm water into the colon before the lamp is introduced.

The illuminating power of the small Edison lamp at the end of the diaphane is equal to twenty-five volt ampères, or eight candle-powers. While this method is useful for determining the topography of the duodenum and of the colon, the hopes expressed by Heryng and Reichmann that it might give information concerning the location and origin of doubtful tumors of the abdominal organs has thus far not been realized. It sometimes becomes necessary to distinguish between the transilluminated area of the stomach and that of the transverse colon, for occasionally the transverse colon will be illuminated if it is distended by gases and not by fecal matter when the diaphane is in the stomach, and, vice versâ, the distended stomach will be illuminated at times when the light is really in the transverse colon. In order to separate one of these organs from the other in diaphany, I had recourse to a method by which the organ which I wished to exclude from the illumination was filled by a liquid sufficiently dark and opaque to cut off the rays from the lamp after its introduction. For this purpose I used a soup made of bouillon thickened with brown flour or the juice of huckleberries, of which sufficient is added to water to give it a deep color. In instances in which the light was in the transverse colon and the stomach prevented exact localization of the large bowel by participating in the reflection of the transilluminated area, I could observe a distinct reduction in the light area from the epigastrium and left hypochondrium downward toward the location of the transverse colon, when the patient would slowly drink one pint to a pint and a half of this soup or the same quantity of huckleberry lemonade. In other cases I noticed that the transilluminated area would almost entirely disappear when the stomach was filled in this manner, although the light was in the colon. These patients, on having their stomachs distended later on with CO_2, were shown to have gastroptosis, their stomachs intervening between the lamp in the colon and the observer. In case the light is introduced into the stomach and we wish to exclude the colon from participating in the reflection of the light, the obstructing liquid must be slowly injected into the colon, or allowed to run in, according to Hegar's method.

In cases of gastroptosis it is at times of utility to illuminate the stomach on one day, and thereafter distend it by air or CO_2, mapping out its position on the abdomen, and on the following day illuminate the colon, distend it with CO_2, and map out its position in a similar manner. Unless the patient has become quite accustomed to the use of the stomach-tube, colon sounding, and irrigation, it is not advisable to determine the location of both colon and stomach on the same day in the method described, because great nervous tension, irritability, and apprehensiveness will seriously interfere with the proper execution of this plan. If the stomach or the colon has been filled with a dark liquid to obstruct the light, either of these steps will prevent the further transillumination of that particular organ on that day. It is well to wash out the colon or the stomach with water before each transillumination. While I am skeptical as regards the value of the method for the diagnosis of tumors, I have had sufficient experience with it to recommend it as a method for locating the colon, and at the same time proving its permeability, for the diaphane is at the same time an illuminator and a sound.

The Use of the X-Rays, or Röntgen Rays, in Diagnosis of Intestinal Disease.

So far the Röntgen rays have offered no very decided aid for the diagnosis of intestinal diseases. The reason for this is to be sought in the fact that the X-rays can give information only concerning conditions in the abdomen which will obstruct the course of the rays and produce a shadow on the fluoroscope. If, therefore, a method can be devised by which portions of the human intestines can be made impenetrable to the Röntgen rays, their normal or abnormal location may possibly be demonstrated by this method. In case of the human stomach, this has been utilized by coating the inner gastric walls with a thin layer of bismuth subnitrate, which cuts off the X-rays. For this purpose the intragastric powder-blower is an available instrument. The course and location of the human colon can be demonstrated in the same manner, for it is possible to coat the inner wall of the colon by bismuth subnitrate through a long Kuhn or Langdon tube introduced through the rectum. Previous to this the colon must be washed out thoroughly. The normal feces are not recognizable by means of the fluoroscope, but if they contain any metallic salt, such as bismuth subnitrate or chlorid of iron, they can be recognized. I have convinced myself that it is possible to render displacements of the colon visible to the naked eye by the X-rays, in the method described. Metallic sounds introduced through the rectum can also be recognized by the fluoroscope. However, as these abnormal positions can be equally recognized by other less complicated and less expensive methods, I have my doubts whether the Röntgen rays will ever become extensively employed as a diagnostic method outside of hospitals.

Boas and Levy-Dorn ("Deutsch. med. Wochenschr.," 1898, No. 2) have proposed a method of recognizing strictures of the gastro-intestinal canal by skiagraphy. Capsules coated with an insoluble material and filled with bismuth subnitrate cut off the X-rays, and their location can be recognized in the intestines, but I doubt very much whether this method will exactly locate any stenosis, because the intestine is not a simple tube, but a labyrinth of convolutions superimposed upon each other, so that even if the bismuth capsule could be recognized, it is absolutely impossible to state whether it is in the stomach or intestines, and in what convolution of the intestine it is located. Metallic bodies like a Murphy button produce a marked shadow on the fluoroscope. Boas ("Diagnostik u. Therap. der Darm-Krankheiten," S. 92) states that he has assured himself that single portions of the colon—for instance, the descending colon and the haustra coli—can be recognized by this method. As the kidneys can be distinctly recognized on the fluoroscope, a differential diagnosis between tumors of the kidneys and those of the intestine, liver, or spleen might, in favorable cases, be made possible by this method.

Palpation and Percussion of the Abdomen.

The success of palpation depends very much upon the tact and skill with which it is carried out. There are few clinical diagnostic methods that permit of a clearer insight into the experience and resourcefulness of a clinician than this simple procedure. Palpation should be carried out both with the patient in the dorsal recumbent position, and with knees drawn up, and also the erect position. If the abdominal walls are very fat or the muscles tense, the patient must be directed to breathe regularly and very deeply. Some patients require to be placed in a hot bath and to be palpated in it in order to relax the abdominal muscles. There are two methods of palpating—

one with the tips of the fingers, and the other using the entire palm of the hand. It is well to begin with the palm of the hand placed flat over the abdominal surface and slightly bent. It is bad practice to place the hand perpendicularly and dig down into the abdomen with the finger-tips. The palpation should be carried out as tenderly and gently as possible. Many beginners feel nothing because they press too hard. The method can not be learned by describing it; it can be learned only at the bedside. The first thing to find out by palpation is the possible existence of painful areas, and whether they are diffuse or circumscribed. One should not ask the patient whether a certain pressure gives pain, but simply observe the features while the process of palpation is being carried out. Any real painfulness will, on the slightest pressure, cause an expression of suffering and distortion of the features. The exact location of the pain must be noted.

There is one factor which may lead to a mistaken diagnosis in palpation, and that is the confounding of the pancreas with a tumor. Normally the pancreas can not be felt through the abdominal walls; but if these are relaxed and very emaciated and the stomach is empty, then the pancreas can be felt occasionally in the epigastric region, and has been mistaken for a tumor. In gastroptosis, or downward displacement of the stomach, the pancreas becomes more exposed above the lesser curvature. It can be easily determined that it does not belong to the stomach when the latter is distended by gas. Fecal tumors in the transverse colon may simulate tumors of the stomach. If there is any doubt, a laxative or large enema should be given to clear out the colon.

The more practised and skilful the examining hand of the physician, and the thinner and more yielding the abdominal walls are, the more easily the fingers will penetrate the abdomen when palpating, and the better are the results of this procedure. Palpation of the abdomen is very difficult, however, when sensitive, ticklish patients arbitrarily contract their abdominal muscles, or when the abdominal walls are somewhat distended as a result of ascites, meteorism, and larger abdominal tumors; it is absolutely impossible in the case of advanced edema of the abdominal walls and with ascites. If a relaxation of the abdominal muscles in nervous patients can not be brought about after using all possible artifices, anesthesia may become necessary. If the tension of the abdominal walls is caused by ascites alone, it must be artificially reduced. The conditions for a successful palpation through the relaxed abdominal walls are most favorable just after the drawing off of the liquid, whereas if one waits until the next day, the tension may again have reached an advanced stage as a result of a new collection of ascitic fluid. In meteorism it is advisable to empty, if possible, the intestines, by means of clysters and aperients; but even when meteorism is absent, the palpation of the abdomen is best made after an abundant evacuation or on an empty stomach, in the morning.

Method of Palpation.—Palpation, to be most instructive, should be carried out when the patient is lying on his back, with his head slightly raised, so that the expression of his countenance may be observed. The patient should bend the knees and draw them up, so that the greatest possible relaxation of the abdominal muscles can be attained. If the persons are very timid, excitable, and sensitive, it is advisable to draw their attention away from the examination by making them interested in some topic or affair totally extraneous to the examination they are undergoing. The mouth should always be kept open, in order to prevent, as far as possible, all arbitrary contractions of the abdominal muscles. Since deep inhalation is followed by deep exhalation, during which the abdominal muscles are naturally relaxed, the patient should be asked to breathe deeply, but quietly. The investigation should always be made with warmed hands, because cold may easily cause reflex contractions of the abdominal muscles. Among the first things to determine by means of palpation is the condition of the abdominal walls and of the contents of the abdomen. Even in superficial palpation one can usually determine whether the abdominal walls are very adipose or not, and whether they are infiltrated by edema. In case the latter condition exists, the abdominal walls have a doughy consistency, and when the fingers are pressed into the abdomen, they leave an impression lasting for some time. The tension of the abdominal walls must also be noticed—whether it has increased or is normal. The abdomen of healthy people shows, during palpation, a uniform, soft, slightly elastic resistance, while in advanced meteorism it shows a resistance like an air-cushion. In the case of emphysema of the abdominal walls, which occurs very rarely, a distinct crackling is heard under pressure. The next duty will be to examine the localities where herniæ are most liable to occur. The permeability or occlusion of the hernial orifices should be ascertained, etc.; if a hernia exists, it should be determined whether it is reducible or not. In order to reach a sure result concerning the condition of the organs directly below the abdominal walls and also of the deeper abdominal organs, the following procedure is recommended: One or both hands are laid upon the abdominal wall. Then one tries gradually to penetrate downward, with a rotatory motion and without removing the flat of the hand from the abdomen, gradually increasing the pressure. The examination is rendered difficult or impossible by a too rapid procedure, because the patient, especially if his abdominal walls are sensitive to pressure, reacts against it by energetically contracting his abdominal muscles, so that nothing can be felt through them. During the examination the expression of the patient's face should always be watched, so that the sensitiveness to pressure located at the several places in the abdomen may be recognized.

Pain spreading over the entire abdomen and much increased, even by slight pressure, may indicate acute enteritis, peritonitis, or colitis. Pain caused by pressure in the right hypochondrium in the region of the cecum is suggestive of typhoid, or, in rarer cases, of tuberculous ulcers; if, at the same time, a strong resistance can be made out in this neighborhood, it suggests appendicitis, typhlitis, or perityphlitis. Sensitiveness to pressure at other places of the abdomen also occurs in the case of malignant tumors of the intestines, as well as in axis torsions or rotations, invaginations, and stenoses. Peritonitic friction, which can often be detected in perihepatitis and perisplenitis, is but rarely observed in perityphlitic abscesses and intestinal ulcers. Perihepatic and perisplenic friction sounds are produced by breathing, but respiration causes no friction sounds between the intestines and abdominal wall in case of appendicitis, perityphlitis, and intestinal tumors. These sounds, if they can be heard at all, are caused only by voluntary motions of the patients or by displacement of the abdominal walls on the part of the examiner, and only exceptionally by intestinal peristalsis.

Edebohl's method of palpating the appendix is given in the chapter on Appendicitis.

The sensation of *fluctuation* is imparted to the palpating hands by undulatory (wave-like) motions caused in any liquid that may be contained in the abdomen in considerable quantity. The motions are started by short taps or beats on the external abdominal wall. It is observed in ascites, large abdominal cysts, and at times in connection with periappendicial abscesses.

Malignant tumors, advanced chronic infiltration of the intestinal walls, coproliths, and cysts of the omentum require much patient palpating, and then they can often be recognized only as an ill-defined resistance in the abdomen, not permitting of exact diagnosis by palpation alone.

Tumors of the Intestines (Carcinoma and Sarcoma).—The differentiation of gastric neoplasms from those of other abdominal organs has been detailed in the author's work on "Diseases of the Stomach," second edition, page 556. Malignant neoplasms are found much more frequently in the large than in the small intestine. Usually they are distinguished by great hardness and by uneven or lumpy surface. They are usually sensitive to pressure. So long as they have not grown together with other parts, they show some degree of mobility. They show very slight respiratory mobility. Their detection is often rendered easier by the fact that they cause a stenosis of the intestines, which at first produces a dilation of the section of the intestine which lies above the stricture. This enlargement may be detected by palpation, percussion, and often even by inspection. When the stenosis is absolute, the abdomen is uniformly distended as a result of the general meteorism (see chapter on Obstructions).

Fecal Tumors (Coproliths).—These have already been considered in the chapter on Constipation. Their recognition and differentiation from malignant tumors are easy whenever a gradual change in position downward can be determined in the tumor, when they are not painfully sensitive to pressure, when they may be partly divided or pressed in under great pressure, and also when they become smaller or entirely disappear after the use of aperients. Their detection and differentiation from malignant tumors are rendered very difficult if they are of considerable size and have acquired great hardness from exsiccation, but especially when they have produced secondary inflammation and local peritonitis, whereby they become sensitive to pressure. In such cases of obstruction by coproliths the utilization of the food is seriously affected by accompanying digestive disturbances. Coproliths have been described which presented a tortuous central canal through their mass, so that, in spite of purgative drugs and of repeated evacuations, no sensible diminution in their size was noticed. In other cases the fecal masses had collected in the haustra or in a diverticulum, so that the central passage remained open. Such types are very difficult to diagnose. The fact that malignant tumors also cause large massing of feces renders the differential diagnosis more difficult. In such cases the fecal tumors can be detected only after long observation ; indeed, sometimes not until after death. The only reliable criterion during life is the influence of aperients and purgative enemata upon the configuration of the tumor. It has been claimed (Pick, Fleischer) that spastically contracted intestinal loops and adhesions between the intestines and abdominal wall can be detected by palpation. Although I have observed numerous cases where cicatricial strands existed as a result of stenosis of the intestine, I have not been able to diagnose them by palpation, as a rule, even though the abdominal wall was thin and relaxed. The cases occurring under my observation suffered considerably from pain, which, though not present at all times, was readily started up by palpation.

The changes in form of the abdomen occurring in acute idiopathic dilation of the colon are described in a separate chapter.

Atony of the colon or compression or stenosis of this organ is occasionally accompanied by marked distention, particularly if the descending portion is obstructed ; in those cases the expansion of the transverse colon can often be recognized by inspection.

Percussion.

Percussion with the finger is, as a rule, preferable to the pleximeter finger percussion and to pleximeter hammer percussion, because it allows one to estimate the resistance which the organs have under the percussing finger. Only occasionally, in the case of an excessive accumulation of fat in the abdominal walls, will the pleximeter hammer percussion become necessary. When percussion of the intestinal loops filled with gases is executed, a tympanitic sound is heard. This sound is much deeper over the large than over the small intestine, by reason of its greater volume, whereas the intestine filled with liquid or solid matter gives rise to a muffled sound. In the case of intestinal meteorism the sound is much deeper than the normal, on account of the increase in volume of the intestinal loops, and by this deep note an advanced distention of the large intestine resulting from a stricture may be detected by means of percussion. If the inflation of the intestines, and therefore also the tension of the abdominal walls, is very great, the sound is of a dull, tympanitic character, or even muffled. While in meteorism of the intestines the percussion sound varies in the different places of the abdomen, as can easily be recognized, in peritoneal meteorism it is everywhere the same, and the hepatic and splenic dullness having disappeared, as a rule, neoplasms of the intestines or fecal tumors that have attained considerable size give rise to a dull sound on percussion, and the same also applies to perityphlitic and appendicial abscesses. It is, however, advisable, during gentle percussion over them, to penetrate further with the finger or pleximeter if the sound is tympanitic, because loops of the smaller intestine filled with gas often lie between the abdominal wall and the district of the muffled sound, and must first be pushed out of the way. According to Leo ("Diagnostik der Krankh. der Bauchorgane," second edition, p. 109), the percussion resonance of the left hypochondrium is of a duller tympanitic character than that of the right corresponding region, which he attributes to the presence of consistent fecal matter in the descending colon and sigmoid.

Mensuration.

Since the circumference of the abdomen is subject to very great individual variations and is very much affected by the amount of food taken in and by the evacuations, measurements with a tape-measure are of use only in determining the increase or decrease in individual cases under similar conditions. In such measurements it is a matter of routine to take the distance from the xiphoid process to the navel, from the navel to the symphysis, the distance between the anterior superior spine of the ilium and the circumference of the abdomen at the navel. If one wishes to be accurate about the increase or decrease in the abdominal circumference in the case of ascites, it is advisable to mark the points in question with a pencil of indelible ink (soluble blue), and to measure with a steel band.

Auscultation.

The value of auscultation as a diagnostic method in intestinal diseases is not considerable. I have already referred to the occurrence of perceptible frictional noises which may be heard with the stethoscope. A bubbling noise may often be heard by moderate pressure on the abdomen in the ileocecal region. In former times these noises were considered pathognomonic of typhoid fever. They are, however, heard also in other diseases of the intestines. In the case of exaggerated peristalsis, very many kinds of noises (borborygmi) are heard during digestion. If they arise in the small intestine, their resonance is higher than in the colon, but these sounds are of no value whatever in diagnosis. Localized friction sounds synchronous with the respiratory movements are occasionally audible in diaphragmatic peritonitis.

Exploratory Puncture of the Abdomen.

This is rarely used except for ascertaining the nature of any disease that has involved the peritoneum. Nothing is required for this purpose except a rather large sterilized hypodermic syringe, the needle of which must be at least eight centimeters long. The skin through which each puncture is made must be thoroughly sterilized before each puncture. The intestines, owing to the air and gas they contain, occasionally float to the top of the ascitic fluid, and lie close under the abdominal walls. But if the patient be placed in a sitting or erect attitude, and the puncture be made below the umbilicus, there is no danger of injuring the bowel.

By this method Dock and Riedel (cited by Dock, "Amer. Jour. of the Med. Sci.," June, 1897, p. 655) discovered carcinoma (in Riedel's case probably a mixed tumor—sarcoma carcinomatodes) of the pleura and peritoneum respectively. They found a large number of asymmetrical (pathological) mitoses and cells with disrupted chromosomes in the liquid withdrawn by aspiration. Upon this evidence they based the diagnosis. When no liquid effusion is contained in the peritoneum, exploratory puncture is connected with more or less danger, owing to the greater possibility of injuring the intestines, because an effusion can be made to separate the intestines from the abdominal walls by bringing the patient into proper position.

In a case of peritonitis due to carcinoma von Leyden found in the ascitic fluid gained by puncture a peculiar ameboid rhizopod which Schaudin designated "Leydenia gemmispara" (von Leyden u. Schaudin, "Sitzungsbericht d. königl. preuss. Akad. d. Wissenschaft," Berlin, Bd. XXXIX, 1896). The same protozoon was found at Nothnagel's clinic and also by Lauenstein in ascitic fluid of peritoneal carcinoma; Dr. Harvey L. Gaylord, of Buffalo, has described a very similar organism as the cause of carcinoma. There is, however, no satisfactory evidence at present in favor of the view that this characteristic ameboid organism stands in etiological relation to the coexisting carcinomatous disease (Nothnagel, *l. c.*, p. 782; also Hugo Ribbert, "Lehrbuch d. allg. Pathol.," S. 614, Leipzig, 1901).

Direct puncture of the intestines has been emphasized as a remedial measure for the relief of the tremendous meteorism occasionally observed in cases of intestinal occlusions and stenoses. In my opinion the method is not free from danger, and should be used only after all other palliative measures have been exhausted and permission for operation has been refused. Curschman, who has warmly advocated this procedure, claims to have employed it frequently and to have produced a direct cure in three cases of intestinal axis torsion or volvulus thereby (Curschman, "Achsendrehung," "Deutsch. med. Wochenschr.," 1883, No. 17, also *ibid.*, 1887, No. 21).

I will refer to this method again in the consideration of the treatment of intestinal obstruction, but must even here caution against its employment excepting under the limitations specified.

The Technic of Exploratory Puncture of the Abdomen.—Although the most frequent indication for this is ascites, I have deemed it advisable to consider this method here because it is available for the diagnosis of diseases of the peritoneum. Before the puncture is made we must ascertain that there is distinct dullness on percussion and a feeling of fluctuation on palpation in the semierect position at the place at which the puncture is to be made. I advise against puncturing, at all, tumors in the following organs: Liver, kidney, spleen, bladder, and uterus. The spot to select is the linea alba, midway between the umbilicus and the symphysis, because the danger of injuring a blood-vessel is least at this spot. If the linea alba for any reason is not available, the next safest spot is midway between the anterior superior spine of the ilium and the navel. Strict asepsis is necessary: the skin, the instrument, and the hands of the operator should be sterilized. Penzoldt advises plunging the needle or trocar through the blunt end of a sterile rubber condom, and, after it has entered the abdomen, to tie it over the cannula. As the guide is withdrawn the condom prevents the entrance of air into the abdominal cavity, and can also be utilized to conduct the outflowing serum into a proper vessel. It will prove expedient to consider the increase in thickness of the abdominal wall, which is sometimes caused by the anasarca, and to apply the index-finger about from four to five cubic centimeters (about 1¾ to 2 inches) from the point of the needle or trocar. The puncture must be done perpendicularly to the skin; with a very slight rotatory motion, a sudden, energetic stab is made, so calculated that it is immediately arrested on penetration through the abdominal wall. When it is done for the relief of meteorism, a hollow needle should be employed that is closed by a stop-cock. The end having the stop-cock is attached to a rubber tube, and this is conducted into a bottle filled with water, which is inverted in a basin also filled with water, so that the escaping gas can be caught and measured in the inverted bottle after the puncture has been made and the stop-cock opened. For the relief of meteorism the needle, after disinfection, is plunged into one of the most expanded intestinal loops in the manner described. Sometimes the puncture must be repeated on several different loops, and at other times, although the abdomen seems immensely distended, puncture is not followed by the desired result. If there is any evidence of intestinal paralysis or gangrene, the procedure must not be undertaken. The manner in which the method produces its effect, however, is analogous to the manner in which gastric lavage works in these cases. The evacuation of gas brings about an equalization of the tension and such a reduction of the pressure within the abdomen that a freer movability of the intestine, which is held tight by its own tension, becomes possible. By this its liberation from incarceration is facilitated.

The dangers attached to the procedure arise from the changes which the intestinal wall may have undergone by the prolonged tension prior to the puncture. An intestinal loop that is excessively dilated in this manner may readily tear if its wall has become in the least degree gangrenous. Under no circumstances should one hold the needle tightly in the fingers after the puncture is made, but it should be allowed freely to follow the movements which the intestine happens to execute. If the musculature of the intestinal loop that has been punctured is still contractile, the small opening will promptly close; but if the loop is paralytic, closure will not take place, and the exit of gases and liquid feces into the peritoneum may occur. Unfavorable cases have been reported by Hoffmann, Fraentzel, Fürbringer ("Cong. f. innere Med.," 1889), also by Körte ("Berlin. Klinik," No. 36, 15). The dangers of puncturing abdominal cysts are also not sufficiently considered in the text-books. They should be especially emphasized in the case of pancreatic cysts (see Hemmeter and Adler, "A Chemical, Physiological, and Clinical Study of Pancreatic Cyst Fluid," "New York Medical Record," August 6, 1898).

It might be presumed that when a pancreatic cyst is tapped under strictly antiseptic precautions, no harm could ensue even if some of the fluid escaped into the peritoneum. This view was held by Senn, but the experiments of Hildebrand and Rosenbach-Jung have demonstrated that escape of pancreatic fluid into the peritoneal cavity may produce fat necrosis. Rosenthal, Cathcart, and Tremaine have reported cases in which the entrance of cystic fluid into the peritoneal cavity caused collapse and compelled a speedy laparotomy. Küster and Kocher reported cases in which the symptoms of peritonitis were produced, and in Socin's case death was attributed to entrance of the cyst fluid into the peritoneal cavity. It is possible that the stomach or colon may be pushed between the pancreatic cyst and the abdominal walls, when adhesions exist between the cyst wall and the stomach. It is impossible to determine to what extent the stomach may be in contact with the anterior abdominal wall, even when it is artificially distended. It is intelligible in such cases how the trocar may be pushed through the stomach or intestines in the efforts to reach the cyst. In Dixon's case the cyst refilled exceedingly rapidly after it had been evacuated by puncture, and several punctures did not produce a cure in Steele's case (Hemmeter and Adler, *l. c.*).

Inflation of the Intestines, for Diagnostic Purposes, with Carbonic Acid Gas, with Air, and by the Injection of Water through the Anus.

Artificial Inflation of the Intestines by Means of Air.—This method of rendering the large intestine more accessible to physical methods of diagnosis is attributed to von Ziemssen. Originally this clinician recommended CO_2 for distention of the colon, but this has been supplanted by distention with atmospheric air. In order to inflate the intestine with air it suffices to introduce an elastic intestinal tube, or, still better, a stiff-walled, thick, rubber tube, as far as possible through the anus, after the intestine has been freed as much as possible from excrement by means of a large irrigation or enema. The outer end of the probe is connected with a double rubber bulb apparatus, such as is used for spraying, or with a pressure pump containing a known quantity of air, which is gradually pumped in until the intestine is sufficiently inflated. If resistance is encountered when the tube is introduced in the rectum, it is advisable to pump in some air or to let some water or oil flow in, so that the fold of the mucous membrane in which the tube is caught may be pushed aside. After the examination has been ended and the rubber bellows or pump removed, the air may be almost entirely removed through the intestinal tube. Massage of the large intestine assists in evacuating the air. If one desires to inflate the intestine, according to the proposal of von Ziemssen, by means of CO_2, an aqueous solution of tartaric acid and sodium bicarbonate must gradually be introduced separately through a funnel. In order to distend the colon in this way twenty grams of sodium bicarbonate and eighteen grams of tartaric acid are necessary. Since the amount of gas necessary for the inflation of the intestines in different persons varies extremely, it requires a longer or shorter time until sufficient CO_2 has developed for the proper distention of the colon. The slow distention by CO_2 in some cases, or the collapse of the colon after it has once been distended by this gas, necessitating new quantities of the foregoing solutions for its generation, has been explained by assuming that the CO_2 is absorbed more rapidly in the intestines than is air. It is not very probable that such large quantities of gas can be absorbed in the very brief periods in which the colon is sometimes observed to collapse. It is more than likely that the gas escapes through the ileocecal valve into the lower portion of the ileum. Rosenbach recommended a procedure by letting gaseous CO_2 flow into the intestine from an iron shell filled with liquid CO_2; Illoway and Schaetter employed for the same purpose an inverted siphon charged with a strongly carbonated liquid. Fleischer holds that the employment of CO_2 has this further disadvantage, that it irritates the intestinal walls. This has not been my experience—I rather think that the means and methods to generate the CO_2, but not the gas itself, may irritate the intestine. The gas from a siphon holding from 1 ½ to 2 liters of carbonated vichy or seltzer will not be sufficient to distend the colon, but distention by air is simple and answers every purpose. The conclusion that CO_2 irritates the intestinal walls is based on the statement made by von Ziemssen and Rosenbach that the CO_2 never penetrates beyond the ileocecal valve, while it is observed by Mamsch that after more than one liter of air has been introduced it penetrates also the small intestine, especially if the introduction was slow and gradual. In my opinion the observations of these clinicians are explainable by the fact that the evolution of CO_2 by the methods employed is very brisk and the rapid distention is followed by reactive colon contractions, whereas the inflation by air being done by hand, must of necessity be more gradual and slow.

The inflation of the large intestine serves, in the first place, for the detection of strictures of the intestines and their location. If the passage of the entire intestine is free; if neither organic strictures nor great massings of feces exist at any place, the large intestine is uniformly and very much distended. This may be determined by percussion, and also by inspection and palpation if the abdominal walls are not too thick. The greatly distended large intestine may thus easily be distinguished from the small intestine, which lies between the ascending, transverse, and descending colon, and which has a high tympanitic sound. In cases of stricture in the lower part of the colon little air can be introduced: the lower the stricture, the smaller the amount of air that can be pumped in. Those parts of the intestine above the stricture are not inflated. If the large intestine be strictured in its upper half, it is inflated only up to that point. Since the sound over the intestine above the stricture does not vary at all, whereas below the stricture it becomes louder and deeper the more air is introduced, the location of the stricture may consequently be determined with some exactness, especially if those parts of the intestines in question are again percussed after the air has been expelled by massage. Displacements of the intestines, and especially of the large intestine, may be recognized much more readily by means of percussion, palpation, and inspection after larger quantities of air have been forced in than before.

The various positions which the transverse colon assumes in enteroptosis can not be demonstrated in a more practical way. Normally the transverse colon is located at a level of from two to four centimeters above the umbilicus in males, along a line drawn transversely across the under border of the costal arches. In females it is normally a little lower—I find it most frequently on a line running through the umbilicus. Of course, the transverse colon is quite movable even normally—I have observed a difference of from three to five centimeters in the same healthy individual when examined on different days and before and after heavy meals. In enteroptosis the transverse colon may assume various shapes, according to the degree or extent of the depression. If the middle of the transverse colon descends, leaving the hepatic and splenic flexures in normal position, the colon as a whole assumes an M shape. If the two flexures before mentioned descend to some extent together, but not so low as the middle part of the transverse colon, the whole large intestine assumes the shape of a V. There is also a V-shaped transverse colon, which arises from a sharp angle in the descended portion, which, as I have occasionally observed, may touch the bladder (see Chap. XXX on Abnormalities in Form and Position).

The differential diagnosis of intestinal tumors and tumors of other organs may also be aided by this inflation method in many cases. After the large intestine is inflated, tumors of the deeper lying organs or parts of organs (kidneys, retroperitoneal gland, spinal column) can no longer be felt, because they are covered by the inflated intestinal loops. It is also stated by Minkowski that abdominal tumors usually move back in the direction where the organ is normally located when the intestines are inflated with air or filled with water. It is for this reason that, for instance, tumors of the pylorus evade detection by means of palpation when the intestine is distended, whereas those of the intestines may become more distinct, especially if the intestinal loop upon which they are situated is pressed against the abdominal wall. In his "Diagnosis of Abdominal Tumors" Osler does not mention inflation of the intestines in the clinical histories of his cases of intestinal tumors, attributing importance, however, to the palpable hardening during contraction of the hypertrophied intestinal wall in the vicinity of the growth, and the bubbling of gas through the tumor, which may be heard as well as felt.

Introduction of Water by the Rectum.

Method.—A thick rubber (Langdon) tube is introduced as far as possible up into the intestine through the anus. The tube is connected with an irrigator on which there is a calibrated indicator, so that the amount of water introduced may be read off at all times. If a part of the water escapes again past the tube during the inflow, it is advisable to make the closure of the intestine tighter, either by means of a cotton wad, which is introduced by the side of the tube, or by digital compression of the sphincter ani. Under normal conditions from three to five liters of lukewarm water can pass into the intestine with a certain rapidity. The rapidity is increased if the patient gets on his knees and elbows, because in this case the pressure in the abdomen becomes negative. If a stricture in the lower part of the intestines exists, but very little water passes in; if it is located in the upper half of the large intestine, the flow of the water is checked after the introduction of one, one and a half, or two liters of water. This method may also be used for the detection of dislocations of the intestines. It does not yield so accurate results as the inflation of the intestines by means of air. The utility of this method is considerably lessened by the fact that in many patients, and especially in those whose intestines are very sensitive, considerable abdominal distress arises after one or one and a half liters of water have entered, so that the examination must be discontinued. Distention of the intestines by water is sometimes unavoidable if the electrodiaphane is to be used in the colon, in order to prevent the Edison lamp from becoming too hot, and also when it is desired to test the capacity and tonicity of the colon.

CHAPTER IX.

DIAGNOSTIC SIGNIFICANCE OF EXAMINATION OF FECES AND URINE.

BY HARRY ADLER, A. B., M. D.,

ASSOCIATE PROFESSOR OF THE DISEASES OF THE STOMACH AND CLINICAL PATHOLOGY,
MEDICAL DEPARTMENT, UNIVERSITY OF MARYLAND.

FECES.

The study of the feces bears the same relation to the study of intestinal derangements as the examination of the urine does to the diagnosis of renal diseases or the examination of the sputum to respiratory affections; and yet this study has always been, and is even now, in this era of careful investigation of diseased conditions, restricted to a comparatively few physicians who make the intestinal diseases their special study. The cause is patent, and lies not only in the unpleasantness of the work and the difficulty of transportation of the feces, owing to their odor, but also to the lack of conclusive clinical data gained by the examination: such data as are gained, for example, by the simple methods of urinary analysis. In intestinal diseases it is often only by systematic and repeated examinations that a positive conclusion regarding the nature of the trouble can be reached; and it is only by such examinations that our knowledge can be enhanced. Too often the practitioner limits himself to inquiring into the condition of the stools, and is satisfied with the replies of the patient or of the attendant regarding the consistency of the stools and the presence of mucus, blood, and pus. Under such conditions a proper understanding of the case is usually impossible, resting, as it does, upon the data of individuals who have not had the training necessary to give a satisfactory report. In the intestinal diseases of infants careful work is being done; and the daily examination of the stools is made a matter of routine by the careful practitioner, and with the happiest results. When the patient is confined to his bed, the stools should be saved for the inspection of the physician. After careful macroscopic examination such portions as are desirable for further study are to be removed and preserved in a well-stoppered bottle. In ambulatory cases glass-stoppered bottles with wide mouths or the ordinary glass salve-box with a screw-top lid may be provided to the patients, in which a portion of the stool can be carried with safety and without inconvenience. The macroscopic examination alone often suffices to make the diagnosis. When it fails, the microscopic examinations should be made and, finally, the chemical analysis.

Boas ("Deutsch. med. Wochenschr.," September 6, 1900)

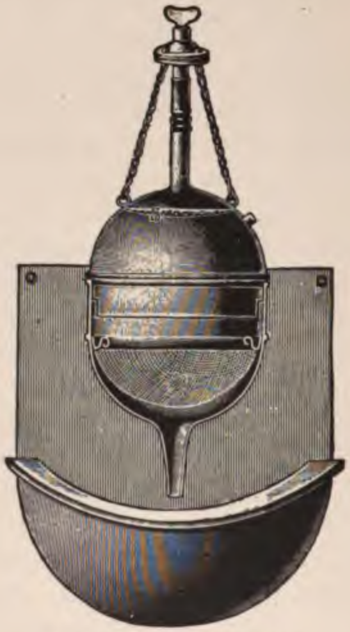


FIG. 14.—BOAS' STOOL-SIEVE.

describes an apparatus of his invention called a stool-sieve, by means of which the stool is thoroughly and conveniently washed, so that undigested remains of food, bits of mucus, concretions, and parasites are readily brought to view and isolated. It consists of two hollow hemispheres of metal, which are brought together in the horizontal plane. The upper hemisphere is provided with a tube, which is connected with running water. The water flows through a fenestrated plate in fine jets, upon a concave sieve placed in the lower hemisphere. This sieve, upon which the feces are placed, has extremely fine meshes, so that the water carries off, through an opening in the lower hemisphere, soluble substances only, while insoluble substances are retained. This procedure requires only from ten to twenty minutes, after which the residue upon the sieve is examined macroscopically and interesting portions are removed for microscopic study. It has the additional advantage that the odor of the stool is in great part removed, so that the examination is less objectionable. By this means the smallest gall concretions may be found, and it is especially useful in examining the stools for the head of a tapeworm and for other intestinal parasites.

General Consideration.

Amount.—The amount of feces passed in the twenty-four hours varies with the quantity and nature of the food taken. With a mixed diet of meat and vegetables from 100 to 250 gm. of feces are passed daily, the average amount being 170 gm. The greater the amount of vegetable food, the greater will be the amount of feces, owing to the large percentage of indigestible (cellulose) matter contained in vegetables. As much as 500 gm. a day may be passed when the diet is purely vegetable. The evacuations do not, however, depend entirely upon the food. The intestinal walls themselves throw off no inconsiderable material. This was well shown by the studies on Cetti, the professional starver. During his fast he had daily evacuations averaging twenty grams in weight. They consisted of epithelial cells from the mucous membrane of the intestines, intestinal juices, salts, cholesterin, bile acids, and bacteria.

Consistency and Form.—The normal feces are passed formed into a cylindrical mass of soft consistency. They are molded in the colon, and often show slight grooves, which are the impress of the folds of the large intestine. The longer the feces remain in the colon, the more water is absorbed and the firmer they become. They may become quite hard and broken into small round masses called scybala. On the other hand, when the peristaltic action is unduly rapid and the feces are hurried through the intestinal tract, time is not given for the absorption of water. The feces are then discharged without form, and have a pulpy or watery consistency. Stools of soft, pulpy consistency, although occurring but once daily, unless the result of purgatives or diet (large amounts of fat or fruit), are always of pathological significance (Nothnagel). The stool is at times passed in the form of slender cylinders, the thickness of a finger or less, or flattened like tape. This may be due to stricture of the rectum, but is by no means diagnostic, as it may occur under normal conditions and often precedes or follows a diarrhea. It may occur while fasting, and has been observed in spastic and paralytic affections of the colon.

Color.—The color is dark brown, due to the presence of converted bile pigment. According to Vierordt, the color is due mainly to urobilin (hydrobilirubin), of which about 0.36 gm. is passed out in the twenty-four hours. Unchanged bile, such as will give Gmelin's test, is never found in the normal feces; its presence is always of pathological significance. Investigations of Frerichs and Nothnagel, made on the contents of the intestines of individuals who have died with no structural or functional trouble of the digestive tract, show that bile pigment is but very seldom found even in the ascending colon, and is usually present not lower than the end of the ileum. When no altered bile (hydrobilirubin) is present, the stool has a grayish or clay color (acholic stool). This same appearance, as will be seen later, may be due to the presence of a large amount of fat. The presence of blood causes the feces to become discolored, varying from a light red to a tarry black. The latter is due to the conversion of the hemoglobin into hematin. The color is influenced by the nature of the food taken: it is dark after a meat diet and lightest after a milk diet. Various *drugs* influence the color of the stools: iron and manganese give a very dark, almost black, color; bismuth gives a grayish black. These discolorations were formerly thought to be due to the presence of the sulphids of the metals taken; but Quincke has shown that it is the oxids of iron and bismuth that are formed. His investigations have been confirmed by Boas. Calomel often gives a greenish tinge, especially noticeable in the stools of infants. This is probably due to a part of the bile pigment not undergoing the usual conversion into urobilin. Rhubarb and senna give a distinct yellowish tinge, due to the presence of chrysophanic acid. Santonin also discolors the feces, as well as the urine, yellow. This, according to Hoppe-Seyler, however, is not due to chrysophanic acid, but to a substance the nature of which is not known. After taking methylene-blue the feces take on a bluish-green color on standing in the air.

Odor.—The odor of the feces, which is characteristic, penetrating, and persistent, is due mainly to the presence of skatol (C_9H_9N), and to a less degree to indol (C_8H_7N). Both compounds are produced by the decomposition of proteids by bacteria. The greater portion of the indol is absorbed by the intestines and appears in the urine as indican (potassium indoxyl sulphate). Two gases, sulphureted hydrogen and marsh-gas, are generated in the colon and increase the offensiveness of the odor. The longer the fecal matter remains in the intestines, the greater the opportunity given for proteid decomposition, and hence the more penetrating the odor. When the feces are hurried through the intestines, the odor is less and may be absent, as in the rice-water stools of Asiatic cholera. In dysentery, as Quincke and Roos first pointed out with regard to the amebic form, the stools may have a peculiar glue-like odor. Fetid stools occur in cases of ulcerating carcinoma of the colon or rectum.

Macroscopic Examination.

Food Particles.—Macroscopic particles of undigested food are often seen in perfectly normal conditions of the digestive tract. They occur when the food has been improperly prepared, so that it resists the action of the digestive ferments. Thus, grains of corn which, owing to their cellulose covering not being broken, are unaffected; or bits of smoked meats the preparation of which has been such as to render them incapable of digestion, may appear in the feces and are easily recognized by their physical properties. When, however, digestible food that has been properly prepared and masticated appears in macroscopic particles, particularly if it is proteid food, as muscle-fibers and connective tissue, it is always significant of an abnormal condition of the intestines. As will be seen, the presence of even microscopic granules of starch is pathological. It is those who have studied the feces who are best able to appreciate the importance of the proper selection and preparation of food. While some individuals are able to assimilate the most indigestible food, in others they are passed out practically unchanged, and the greatest care is required that the food may be rendered assimilable. The assimilative powers of different individuals vary so greatly that it is difficult to define accurately, with regard to the presence of food in the stools, what is of pathological significance and what is not. There can be no doubt, however, that the presence of macroscopic bits of muscle tissue in the stool after a diet containing meat which has been properly prepared is pathological. Schmidt ("Deutsch. med. Wochenschr.," December, 1899, S. 811) gives 100 gm. of well-done Hamburg beefsteak, and draws the following inference from the stools:

1. The presence of *connective tissue* in the stools after the diet of 100 gm. of meat is an indication of a disturbance in function of the stomach, without permitting any conclusions regarding the nature of the disturbance, whether it be subacidity, anacidity, or hypermotility.

2. If, at the same time, *muscle-fibers* are present, the intestinal digestion is also disturbed.

3. The presence of muscle-fibers without connective tissue indicates a serious disturbance of the intestinal digestion without permitting any deductions, whether it is dependent upon a disturbance of secretion or absorption. The appearance of macroscopic vegetable remains in the feces is of no pathological significance.

Mucus.—The determination of the presence or the absence of mucus in the stools is of the greatest value in the diagnosis of intestinal diseases. Its presence is a distinguishing feature of catarrh of the intestines as opposed to nervous diarrhea. It is secreted as mucin, and its presence may be demonstrated chemically in the normal stool; but macroscopically or microscopically, its presence is always a sign of a pathological condition. The truth of Nothnagel's statement that it is an indication of divergence from the normal is now generally recognized. It is present, however, in some comparatively trivial affections; for example, it may occur as a thin layer over the feces in constipation, owing to the irritation of the hardened feces on the rectal mucous membrane. Again, it is present in the most serious pathological conditions, such as ulcerations. The mucus occurs in several different forms:

1. As a covering for the hardened feces. It may be glassy or cloudy, but is always grayish or white; a brownish yellow color is due to the color of the feces.

2. In masses having the appearance of a membrane, occurring in

strips or forming a more or less perfect mold of the intestines. This is seen in mucous colitis (colitis membranacea).

3. The mucus may be passed in large quantity, so that the entire dejection may be one mass of glassy, jelly-like mucus, with but little or even no fecal matter. This occurs in acute dysentery.

4. As small particles, glassy in appearance, intimately mixed with the feces. If the stool is watery, the mucus may be seen floating around or sticking to the sides of the vessel. If the stool is of a pulpy consistency, stirring it with a glass rod will cause the mucus to adhere to the rod. The particles may be so small as not to be recognizable macroscopically and to require the use of the microscope for their detection. This is seen in catarrh of the small intestine.

5. Nothnagel has described a form in which it occurs as small, yellow particles, varying from a size barely visible to that of a pea, and of the consistency of butter. These bits of yellow mucus may occur very sparsely or in enormous numbers, so that the stool appears as sown with them. They occur very rarely. Boas and Schmidt have never seen them. The yellow tinge is due to bile pigment. According to Nothnagel, their presence indicates catarrh of the small intestine.

The presence of mucus in any of the forms except 1 or 2 is an indication of a catarrhal condition of the intestines. Even in the second form it is at times an expression of a genuine catarrhal inflammation (see chapter on Mucous Colic and Colitis Membranacea). But the converse is not true: the absence of mucus in no way precludes the existence of a catarrhal condition. In atrophic conditions and in catarrh of the upper part of the small intestine, the examination for mucus may be constantly negative.

Blood.—Blood found in the stools is always of pathological significance, whether found macroscopically, microscopically, or chemically. It may be present unaltered, and is then easily recognized, or it may have undergone change, so that its recognition is dependent upon microscopic or chemical tests (see Chemical Analysis of the Feces, p. 241). Blood arising from the lowest portion of the intestinal tract is bright red when passed; when it arises from the small intestine, it is usually decomposed and so altered as to be characterized by a tarry appearance. Profuse hemorrhages from the small intestine, when associated with increased peristalsis, have, however, a red color. The source of the blood is a problem that is to be solved only by the history of the case and the examination of the patient. The possibility of its having a gastric or pulmonary origin must always be kept in mind. Blood-stained mucus is characteristic of dysentery and of ulcerations of the intestines due to any cause. It also occurs in occlusion and tumor of the intestines. *The presence of blood in the stools is always an indication for rectal examination.*

Pus.—When present in large amounts, it is easily recognized by macroscopic examination ; but usually the microscope is necessary to demonstrate its presence, especially when it arises high up in the bowel, as it is then thoroughly mixed with the rest of the stool. When passed pure, it comes from the lowest portion of the bowel, due usually to certain types of ulcers or to the bursting of a neighboring abscess into the gut. It is frequently an accompaniment of mucus and blood, and is then significant of ulceration of the mucous membrane.

Concretions are of several varieties: biliary calculi, pancreatic calculi, and enteroliths. Of these the most important are the *gall-stones.* They are friable yellow or brown masses, having usually smooth surfaces or facets. They may be as small as a millet-seed or as large as a hen's egg. When formed singly in the gall-bladder, they are rounded, but when several lie together, they become angular and faceted. On section a dark nucleus is shown, consisting of bile pigment and calcium salts, surrounded by plates of cholesterin. Nearly all contain cholesterin and bile pigment. Gall-stones consisting of bilirubin and salts of lime and others consisting of calcium carbonate have been described. They are usually small and are very rare. For description of *pancreatic calculi* see chemical portion. *Enteroliths* or intestinal concretions are stony concretions formed in the intestinal canal. They are formed in the cecum, vermiform appendix, and colon ; rarely in the small intestine. Three varieties have been described by Leichtenstern (Ziemssen's "Cyclopedia"):

1. Stony concretions, showing on section successive layers of white, yellow, and brown. They consist of magnesium phosphate, ammoniomagnesium phosphate, and organic matters; and frequently

contain some foreign body, as a fruit-seed or fragment of bone, as a nucleus. They are rounded and rarely exceed the size of a chestnut.

2. Concretions of decidedly less firm consistency, often somewhat elastic. They are made up of indigestible husks of vegetable refuse intermingled with indurated feces and earthy salts, such as calcium phosphate. They may attain the size of an orange.

3. Stones due to the long-continued use of insoluble salts, as chalk, bismuth, and magnesia. These large concretions may give rise to partial or complete obstruction of the bowels. They are most apt to occur in those taking a largely vegetable diet.

Growths.—Fragments of *growths* are extremely important from a diagnostic standpoint, but are very rarely found in the stools. When found, they should be washed, hardened, sectioned, and mounted for microscopic study.

Microscopic Examination.

For microscopic study a minute portion of the stool is placed upon a glass slide, rubbed up with a few drops of water, and covered with a cover-glass. If the stool is watery, a drop or two is simply placed upon the slide and covered with the cover-glass. A solution of thymol or a weak solution of formaldehyd may be added to the stool to make the odor less disagreeable. These additions are, however, not advisable, as they may interfere with the examination. Boas' stool-sieve greatly facilitates the examination. The stool is examined for the presence of *food particles, crystals, mucus, blood, pus, epithelial cells, foreign particles, and micro-organisms, fragments of intestinal tissue, or neoplasms.*

Under food particles are embraced muscle-fibers, connective tissue, starch, and fat.

Muscle-fibers are usually seen in the healthy stools of meat-consumers. They appear as broken yellowish cylinders, often showing the characteristic cross-striping.

When this cross-striping is absent, they have very much the same appearance as waxy casts. Not infrequently they have a greenish-yellow hue, due to being stained with bile. In diabetes with a preponderating meat diet, and in febrile conditions where the proteid digestion suffers, large numbers of muscle-fibers are present; also where there is increased peristalsis they are found, especially when associated with fever, as in typhoid fever. Here we have an additional reason for withdrawing the meat from the diet. From a diagnostic standpoint little is to be gained by the finding of muscle-fibers in the stools. Nothnagel expresses himself as follows: The probable diagnosis of catarrh of the *small* intestine is to be made in cases where no fever exists, but symptoms of catarrh of the bowel (as shown by the presence of mucus) and abnormal quantities of muscle-fibers are found in the stools. The presence of these fibers in abnormal quantities in pathological conditions is an indication for withdrawing or restricting the diet in this particular. Together with muscle-fibers, elastic fibers and bundles of connective tissue are often seen.

Fat occurs in the stool in three forms: (1) As fat droplets; (2) as fatty acid crystals; (3) as crystals of soap. The fat droplets are easily recognized by their being round, slightly yellow, and glistening. They stain black with osmic acid and red with soudan III. The fatty acids occur as slender refractive needles, often in clusters. They are dissolved by the application of heat and are soluble in ether. Soap crystals also occur as needles radiating from a center, but they are not dissolved by heat. They are compounds of Ca and Mg with the higher fatty acids. These forms may be present normally. The ability of the intestines to digest fat is limited, and when it has been taken in large quantities, it is found in the stools. Abnormal amounts are seen in cases of enteritis, diseases of the pancreas, and any condition associated with severe icterus. Acholic stools show very large amounts of fat.

Starch is practically never seen in the normal stool in the form of granules, as found in the stomach-contents. Their presence, even in moderate amount, is *always* pathological. They are recognized by their oval shape and concentric striations and by their staining blue when treated with a dilute iodin solution. Nothnagel says that, as the result of thousandfold examinations, he is able to assert that starch particles are not found in the stools of healthy individuals, even though they have eaten considerable amounts of bread. Plant cells and the cellulose remains of vegetables are to be found in every stool after a vegetable or a mixed diet, but they do not show the presence of starch when treated with iodin. They occur in various forms, but they are easily distinguished by their size, often by their spiral shape, and at times by the chlorophyl contained in them.

Starch granules are found in diarrheic stools and in cases of hyperacidity of the stomach. The absence of pancreatic juice and bile secretions seems to cause no starch to appear in the feces, the starch being converted by bacteria and the diastase of the intestinal juice and bile.

Casein.—Flocculi of casein are often found under pathological conditions when milk is the main form of diet. They do not appear normally, and indicate faulty intestinal digestion. They are often stained with bile and contain fat droplets. Their presence is especially marked in the intestinal indigestion of infants.

Crystals.—Various organic and inorganic crystals are found in both the normal and abnormal feces. They have as yet no clinical significance.

Triple phosphate crystals occur very commonly. They have the same coffin-lid appearance as seen in the urine. They are colorless, rarely tinged with bile, and are dissolved by dilute acids.

Calcium phosphate crystals occur frequently as slender wedge-shaped crystals, radiating with their pointed ends from a common center. They are never tinged with bile, are found in the normal stool, and have no clinical significance.

At times a form of calcium salt is seen as small, yellow, bile-stained round or oval masses. They are found both in normal and abnormal feces.

Oxalate of calcium occurs as small crystals, with the well-known envelop appearance. They are found especially after eating such vegetables as rhubarb, spinach, and radishes, which are rich in oxalates. Fatty acid crystals have been previously described.

Charcot-Leyden crystals having the same appearance as those found in the sputum after an asthmatic attack are sometimes met with in the stools. They have been of interest since Leichtenstern pointed out that they are present whenever parasites inhabit the intestines. He has, on postmortem examinations, been able to find these crystals embedded in the mucus in the neighborhood of the parasites, and considers their formation to be due to the presence of the parasites. He attributes, therefore, great diagnostic importance to them. Their absence, however, does not preclude the existence of parasites. Leichtenstern's assertions as yet lack confirmation. The crystals have been found in the stools in cases of typhoid fever, dysentery, pulmonary tuberculosis, and rickets. They are to be distinguished from fatty acid crystals by their insolubility in alcohol or ether. They are soluble in dilute acids and alkalis.

Bismuth crystals are found after the administration of any of the salts of the metal. They are irregular rhomboids of dark-brown color. They are not sulphids, but oxids.

Cholesterin crystals are not of common occurrence. They have the appearance of broken triple phosphate crystals, but are distinguished by the fact that they become deep red when treated with concentrated sulphuric acid.

Hematoidin, appearing as ruby-red ill-defined crystals, has been described by von Jaksch as occurring after hemorrhage of the intestines.

Blood.—Red corpuscles are to be seen under the microscope only when the blood comes from the lower portion of the bowels or when there has been a profuse hemorrhage accompanied by increased peristalsis. The examination must be made soon after the evacuation, as the corpuscles undergo rapid disintegration.

Pus is seen as an accumulation of polymorphonuclear leukocytes. In the normal stool a few isolated leukocytes are usually to be found. When present in large numbers, they are due to an ulceration of the intestines or to the bursting of an abscess into the tract.

Mucus does not occur even microscopically in the normal feces. It is recognized under the microscope by its hyaline, thread-like appearance. Its recognition is of great importance, as it indicates one of the forms of enteritis, colitis, or proctitis. In doubtful cases the preparation should be stained with thionin, which stains the mucus a reddish violet, while proteid material is stained blue. Pariser ("Deutsch. med. Wochenschr.," 1893, No. 41) recommends the triple stain of Ehrlich, which colors the mucus green, while albumins are stained red. Epithelial cells are often embedded in the mucus.

Epithelial Cells.—Two forms are met with: pavement and cylindrical epithelium. Pavement epithelial cells come from the anal orifice, and are seen but rarely. They are most liable to be found in the mucus covering a firm, constipated stool, in which cases the cells are mechanically stripped off by the passage of the hardened feces. Similar cells have been seen in carcinoma of the rectum. Cylindrical epithelial cells are very common. They occur as the result of desquamation of the epithelium of the mucous membrane, and are most often found embedded in mucus. At times they present a perfectly normal appearance, but oftener they have undergone a fatty or granular degeneration and have lost their nuclei. Goblet cells are occasionally seen. The presence of cylindrical cells in large numbers points to an active desquamation of the mucous membrane epithelium, and their presence in mucus indicates a catarrhal condition. They are most frequently met with in the thin diarrheal stools of catarrhal enteritis, and are also occasionally found in the evacuations in cases of ulcers and neoplasms.

Chemistry of the Feces.

Of the three methods of investigation before mentioned, the chemical examination gives us the least information for the diagnosis of the intestinal disease, although considerable work of this character has been done. In the study of the metabolism the estimation of the amount of nitrogen, fats, and carbohydrates in the feces plays an important part. In the clinical investigations of the functions of the intestines the chemical examinations give us as yet, with few exceptions, but very little insight. Only by persistent work in this line can our knowledge be increased. As the result of numerous examinations, Wehgsarg ("Mikr. und chem. Untersuch. der Feces . . erwachsener Menschen," Giessen, 1853) found the normal feces to consist of:

Water, ... 68.3 to 82.6 per cent.; average, 73.3 per cent.
Solid residue, . . . 17.4 " 31.7 " " 26.7 "

The inorganic substances are chiefly sodium chlorid, sodium sulphate, sodium phosphate, earthy phosphates (about 80 per cent.), iron phosphate, calcium sulphate, and silicic acid (Ender, in "Annal. Chem. et Pharm.," XLIX, p. 335).

Reaction is neutral or, more often, alkaline. Ofttimes the outer portion, which lies in contact with mucous membrane, is alkaline, while the inner portion is acid. This acidity is probably due to acid gases. The degree of alkalinity of acidity may be estimated by stirring up twenty grams of feces with 100 c.c. of water and then titrating with decinormal NaOH or H_2SO_4, using phenolphthalein as an indicator. This work is, however, as yet without value. In typhoid fever the stool is alkaline, but in acute enteritis of children and adults it is acid. According to Wegscheider, the reaction is always acid in feces of infants (Maly's "Jahresbericht," Bd. LXV, S. 182). In some types of jejunal diarrhea the reaction may be amphoteric—*i. e.*, alkaline and acid at the same time—or acid to one indicator and alkaline to another (see Mathes and Marquardsen, "Verhandl. d. XVI. Congresses f. innere Medicin," 1898, S. 360). The fecal contents of the small intestine react alkaline to cochineal, methyl orange, and red lacmoid, and, at the same time, they react acid to phenolphthalein, curcuma, and rosolic acid. If such contents are hurried through the bowel, the feces may show the same reactions as the contents of the small intestine. The alkaline reaction is due to carbonates and phosphates; the acid reaction is due to saturation with carbonic acid, though certain organic acids may, under pathological conditions, augment the acidity.

If a solution of phosphates and carbonates is saturated with carbonic acid gas, it gives the same double reactions to the indicators mentioned as the intestinal contents. The reactions of intestinal contents to various indicators have been studied by B. Moore and T. J. Bergin ("Amer. Jour. of Phys.," vol. III, p. 316). They confirm the results of Mathes and Marquardsen (*l. c.*).

Mucin.—Although, as previously stated, in the normal feces mucus is not to be found in either macroscopic or microscopic examinations, its presence may be easily demonstrated by chemical analysis. For this purpose the feces are mixed with an equal quantity of a solution of $Ca(OH)_2$, and the mixture allowed to stand several hours. It is then filtered, and the filtrate treated with acetic acid in excess. The presence of mucus is shown by the formation of a white cloud.

Albumin.—The feces contain a minute amount of albumin, too small to be detected by the ordinary tests. The amount is increased and occurs as serum albumin in diarrheic stools, in typhoid stools, and whenever there is an admixture of pus or blood in the stool. Von Jaksch has found it in a case of chlorosis. To detect its presence, the stool is thoroughly mixed with water and repeatedly filtered until the filtrate is clear. It is then subjected to the usual tests for albumin—Heller's nitric acid or Purdy's acetic acid and ferrocyanid of potash test.

Albumoses and peptones do not occur in the feces normally. They have been found accompanying albumin in cases of dysentery and when an abscess bursts into the intestines. Von Jaksch found most acholic stools to be rich in peptones. To demonstrate their presence, Hofmeister's, Devoto's, or Salkowski's method may be used. Salkowski's method is most easily carried out, and is quite satisfactory. The clear watery extract of the feces is boiled, and a few drops of acetic acid are added and filtered to remove any albumin that may be present. The filtrate is acidified with five cubic centimeters of HCl, and phosphotungstic acid is added until a precipitate is no longer formed. By careful heating the precipitate collects in one clump. The fluid portion is then poured off, and the precipitate is dissolved in a solution of NaOH. It is then treated with a few drops of a very dilute solution of copper sulphate. If albumoses or peptones are present, a rose-red color is produced (biuret reaction).

Blood.—When unaltered blood has been passed in the stools, it is easily recognized macroscopically and microscopically ; but when, as is often the case, it is disintegrated, it may be so altered that its recognition is difficult and its presence a matter of doubt. In such cases one of the chemical tests or the spectroscopic test is to be made for hemoglobin.

Schönbein's and Almen's Test for Blood.—The feces are first treated with ether and a little acetic acid, to extract the hemoglobin of the blood. To this ether extract old oil of turpentine and a few drops of tincture of guaiacum are added. (Turpentine may be rendered "old" by shaking it up with H_2O_2.) If blood is present, a bluish-green color will be formed on standing for a few minutes.

Teichmann's hemin crystal test is most delicate. A minute portion of the feces in which blood is thought to be present is rubbed on a glass slide with a fragment of salt. It is then covered with a cover-glass, and glacial acetic acid is allowed to run under; the slide is then heated slowly over a small flame until the acid *just* comes to a boil. On examination with the high power of the microscope the characteristic hemin crystals will be seen if blood be present. These crystals are of a brown color and rhombic in shape.

They are insoluble in ether and alcohol, but are easily soluble in a caustic potash solution.

The spectroscopic test may also be used: The hematin is extracted with ether, as in the foregoing tests. This extract is examined for a dark stripe in the red portion of the spectrum, between Fraunhofer's lines, C and D.

Bile Constituents.—That unaltered bile as bilirubin or biliverdin never occurs in the normal stool, but that altered bile in the form of hydrobilirubin gives the color to the feces, has already been dwelt upon. The chemical examination for the presence of bile pigment in pathological conditions is often of the greatest importance. For this purpose *Gmelin's test* frequently suffices. This test consists of adding fuming nitric acid to a small portion of the feces. If bile be present, a rapid transition of colors (red, violet, blue, and finally green) is seen. It is the green color which is significant. This reaction occurs, however, only in thin or mushy stools. The method may be used microscopically, the nitric acid being allowed to run under the cover-glass in contact with the feces. A modification of Gmelin's test which gives very satisfactory results is the following: A portion of the stool is mixed with water until it is quite thin. It is filtered through white filter-paper. The bile is thereby taken up by the paper. It is dried, and a few drops of fuming nitric acid are added. The characteristic play of colors is then easily seen if bile is present.

Schmidt's Test.—This test for both bilirubin and urobilin has been recently recommended by Schorlemmer ("Arch. f. Verdauungs-Krankheiten," Bd. VI, H. 3). It is an excellent method, and is especially well adapted for microscopic examination. A small portion of the stool is thoroughly mixed with a strong solution of mercuric chlorid. After a time, varying from one-quarter of an hour to several hours, the presence of hydrobilirubin is shown by the mixture becoming pink, while the bilirubin is green. A little is placed upon the slide and examined microscopically. If any unaltered bile is present, minute particles of greenish bile-stained mucus, epithelial cells, and muscle-fibers stained with the bile may be seen.

Huppert's is another excellent test, for which see von Jaksch, "Klinische Diagnostik."

Bile acids occur under the same pathological conditions as bile pigments. The most satisfactory test is *Pettenkofer's:* A portion of the feces is extracted with alcohol, the alcohol is evaporated, and the residue dissolved in a weak solution of soda. A small quantity of cane-sugar is added, then concentrated sulphuric acid drop by drop. The presence of bile acids is shown by the appearance of a beautiful purple color.

The formulated sentence of Nothnagel that bile-tinged mucus has its origin in the small intestine has been accepted as dogma and copied from one text-book to another. According to the investigations of Schorlemmer, it also occurs in the affections of the colon alone when there is increased peristalsis. He has arrived at the following conclusions: (1) Large, glassy, macroscopic bits of mucus stained with bile do not have their origin in the small intestine, but arise from the colon. (2) When extremely small bits, barely visible to the naked eye, or exclusively microscopic portions of bile-tinged mucus are found, the suspicion that they arise from the small intestine is justified. This suspicion is strengthened if these bits of mucus occur in thin stools and are thoroughly mixed with the residue of the food. Muscle-fibers, connective tissue, or fatty material stained with bile are not of themselves evidences of catarrh of the small intestine, but when they occur in very large numbers and are associated with microscopic bile-tinged mucus, as just described, they justify the diagnosis. It is not necessary that the stools be watery, although such is usually the case.

Acholic Stools.—When the flow of bile into the duodenum is prevented by any cause, the stools become of a light gray or clay color, and have the consistency of putty. The color is due to the presence of the normal urobilin. The stools show, under the microscope, an abnormal amount of fat. This form of acholic stool is accompanied by icterus and choluria. Stools of the same color may, however, occur under totally different circumstances—viz., when there is no interference with the flow of bile, but when the stool contains an excessively large amount of fat and fatty acids. Why, in this case, the urobilin is not found is not known. According to von Nencki, the bile, instead of being transformed into urobilin, is converted into a colorless substance called leukourobilin. As shown by Bunge ("Jahrbuch d. physiol. u. pathol. Chem.," 1887, S. 192), the brown color may be restored to the feces by dissolving out the fat with ether. We have been able to restore the brown color to the feces by this procedure in cases of acholic stools attended with marked icterus and choluria, and when Schmidt's test showed the absence of urobilin. The acholic stools, when there is an absence of icterus and choluria, have been observed in a variety of conditions: tuberculosis of the bowels, chronic nephritis, chlorosis, fatal cases of scarlatina, and in tubercular peritonitis of children.

Biliary and Pancreatic Calculi.—At times sandy material and calculi are passed without any symptoms indicating their origin. In such cases the determination of their origin by chemical analysis is indicated. Gall-stones consist mainly of cholesterin, bile pigment, and bile acids in combination with lime. Their recognition consists in demonstrating the presence of cholesterin and bile. For this purpose the gall-stone is crushed and treated with alcohol and ether, equal parts, to extract the cholesterin. The alcohol-ether extract is poured off and evaporated in a porcelain dish over a water-bath. The cholesterin is obtained in irregular dentated rhombic tablets. On addition of concentrated H_2SO_4 the crystals become red, then purple-green, and finally yellow. To the residue, after the alcohol-ether extract has been poured off, dilute HCl is added and heated. The addition, now, of chloroform extracts the bile pigment, which is recognized by Gmelin's test.

Pancreatic calculi are characterized by their solubility in chloroform, by the absence of cholesterin, and by the aromatic odor they impart when they are heated (see Minich, "Berlin. klin. Wochenschr.," 1894). Their recognition is of great importance in the diagnosis of obstructions of the pancreatic duct; but, unfortunately, they are but rarely found in these conditions. They are friable, small, and irregular in shape, and are compounds of the carbonate and phosphate of lime.

Unorganized Ferments.—Two methods are made use of to determine the presence of ferments: the fresh feces are thinned with a saturated solution of thymol to prevent the action of bacteria. Now glycerin is added to the feces, which extracts the ferments. The thymol mixture is filtered, and the filtrate examined for ferments, or the glycerin extract is used. Another method, recommended by Leo, is to suspend a piece of fibrin in a watery extract of the stool. After twenty-four hours the fibrin which attracts the ferments is withdrawn, washed, and tested. The following is a method of testing for *trypsin:* the filtrate or the extract is rendered alkaline with sodium bicarbonate, the alkalinity equaling a 1 per cent. soda solution. A bit of fibrin is added and placed in a thermostat at 37° C. for a number of hours. At the end of this time if trypsin be present, the fibrin will have wholly or partly disappeared, and the solution will give the biuret reaction for peptones. To test for *diastase*, a weak solution of starch is added to the alkalinized filtrate and placed in a thermostat for about one hour; at the end of that time the starch will have been sufficiently converted into dextrose to give Fehling's test for sugar if the ferment is present. *Steapsin* may be tested for by adding neutral olive oil to the filtrate, and examining at the end of twenty-four hours with red litmus paper for an acid reaction caused by the formation of fatty acids. These procedures have as yet no clinical value, owing to the lack of knowledge on the subject. Von Jaksch and Leo have found diastase in the normal dejecta of children and adults. Trypsin is not found in the normal stool, according to Leo. Both he and Boas found it in cases of diarrhea. Hemmeter examined the sterile extract of feces from eighteen different individuals for proteolytic, amylolytic, and adipolytic ferments ("Ueber das Vorkommen von proteolytischen, etc., Fermenten im Inhalt des menschlichen Kolons," "Pflüger's Archiv f. d. ges. Physiol.," Bd. LXXXI, S. 151). Four of these were normal individuals, four suffered from diarrhea in consequence of enteritis, and four from atonic obstipation. The feces of the normal individuals always contained some proteolytic ferment acting best in an alkaline medium, and occurring also in the feces of such cases as contained no pepsin in their gastric juice on repeated test-meals. This proteolytic ferment was absent or very faint in patients afflicted with putrid diarrheas. It was absent or very faint in the digestive tests in cases of persistent atonic obstipation. The action of bacteria was excluded in all experiments. No adipolytic ferment was found in the feces in these investigations.

B. Moore and T. J. Bergin have found that the filtered intestinal contents of a dog showed the presence of a proteolytic and diastatic, but not of a fat-splitting, ferment ("Amer. Jour. of Phys.," vol. III, p. 324).

Fermentation of the Feces.—A method for examining the feces has been described by Ad. Schmidt ("Verhandl. d. Cong. f. innere Med.," S. 570), which he says "shall demonstrate disturbances in the functions of the intestines in cases when only general symptoms and subjective complaints point to an insufficiency of the digestive functions and when special symptoms of an intestinal trouble are absent." This method is founded upon the amount of the fermentation of the feces. The patient is given daily 1560 gm. milk, four eggs, three pieces (100 gm.) of Zwieback, a plate of soup of oatmeal (40 gm.), with ten grams of sugar, a plate of flour soup made with twenty-five grams of wheat flour and ten grams of sugar, and one cup of bouillon; 120 gm. of potatoes are also given.

A small amount (0.3 gm.) of carmin is given to color and designate the first stool to be examined. A small portion of the stool is dried to constant weight and weighed. It is then mixed with water and placed in a fermentation tube, and kept at 37° C. Fermentation with the evolution of gases sets in and is divided into an early and a late fermentation.

1. Early fermentation occurs during the first twenty-four or forty-eight hours. Gases are rapidly formed, of which 75 per cent. is carbon dioxid due to the fermentation of starch. The products of albumin fermentation (phenol and indol) are formed either not at all or to a slight extent only. A small amount of marsh-gas and hydrogen sulphid is formed.

2. Late fermentation begins slowly on the second or the third day, and gradually increases, forming gases consisting of CO_2, but only 30 per cent., instead of 75 per cent., as in the previous fermentation, with large amounts of indol, skatol, marsh-gas, and hydrogen sulphid.

In the early fermentation it is the starch that is acted upon, while in the late it is the albuminous cellulose materials. Early fermentation can be considered as present only when in the first twenty-four hours an evident amount of gas is formed. Normally after the diet just described there should be no such fermentation. Its occurrence indicates faulty starch digestion and an abnormal condition of the bowels, especially of the small intestine. Clinically, Schmidt regards only the early fermentation. The late can not be used as a test for the albumin digestion, as it is interfered with by the carbohydrate fermentation. To obtain an idea of the albumin digestion he adds trypsin to the feces, allows it to digest, and then tests for albumoses. In normal stools after adding trypsin very little albumoses are found, but in pathological conditions they occur in considerable quantities. Strassburger confirms his views. Basch has tested Schmidt's and Strassburger's assertion that early fermentation points to a pathological condition of the small intestine and the upper part of the colon. As the result of his work in a large number of cases he has found: (1) That early fermentation occurs in normal cases. (2) Often cases with severe pathological changes in the intestines, as in typhoid fever and atrophic enteritis, show no early fermentation. Work that has been carried out in the clinical laboratory of the University of Maryland gives practically the same results. Stools of normal individuals at times gave as much gas as those of severe cases of gastro-enteritis. Kersberger ("Deutsch. Arch. f. klin. Med.," Bd. LXVIII, H. 5 u. 6) gives numerous objections to the method, and in the "Centralblatt für innere Medizin," November 7, 1900, says: "For the clinic it has no value."* It is evident that in its present state this line of investigation is unsatisfactory and yields no positive information upon which to base a diagnosis. The principle upon which the work is founded seems good, and it is possible that important information concerning starch and proteid digestion in the intestines may, by modifications, be gained by it in the future.

Phenol, Indol, and Skatol.—These substances are formed in the normal process of intestinal digestion, and are to be found in the normal as well as in the pathological stool. They are aromatic substances, and are separated from the feces by distillation. *Phenol*, or carbolic acid (C_6H_5OH), is demonstrated as follows: The stool is distilled and the distillate is rendered alkaline with potassium hydroxid and again distilled. The phenol is thereby freed from the indol and skatol. These substances go over, while the phenol remains behind in the distilling flask. It is now distilled with sulphuric acid. In the distillate phenol can be demonstrated by the

---

*Schmidt, in an article in the "Berlin. med. Wochenschr.," December 17, 1900, modifies his previous statements. He says that his test is especially important in ascertaining the *mild* rather than the severe forms of intestinal disturbances, and, further, that conclusions are to be drawn only from positive reactions. The reaction may be negative in severe cases.

addition of a drop of a solution of ferric chlorid by the formation of an amethyst-blue color.

Indol (C_8H_7N) and skatol (C_9H_9N) are obtained by treating the feces with caustic potash and distilling. They go over in the distillate. Indol gives a red color when treated with fuming nitric acid. Skatol is recognizable by its offensive penetrating odor. It forms colorless platelets, soluble with difficulty in water. It gives a violet-red color with HCl.

Since these substances occur normally, their determination has no clinical significance. It is probable that they are formed in increased quantity under conditions associated with increased putrefaction in the intestines, but we have no satisfactory quantitative methods for estimating the exact amounts formed.

Fats and Proteids.—The quantitative estimation of the amount of fats and proteids in the feces is of great importance in studies of metabolism, but for the diagnosis and treatment of intestinal diseases *per se* it is as yet impracticable on account of the time required to make the determinations. For the estimation of the fats Soxhlet's apparatus, and for the proteids Kjeldahl's method, are best. It is not, however, within the province of this article to describe the technic of these methods, and those interested are referred to von Jaksch, "Klinische Diagnostik," Hoppe-Seyler's "Lehrbuch der chemischen Analyse," and "Pflüger's Archiv," 1899, Bd. XLVI, S. 581.

URINE.

The fact that many disturbances of the function of the intestines are secondary to diseased conditions in other organs calls for a systematic examination in all chronic intestinal diseases, in which must be included the examination of the urine. Thus, for example, a persistent diarrhea may be the most prominent symptom of a chronic nephritis; it is a common accompaniment of amyloid disease of the kidney. These facts are now so well known that no painstaking physician will treat a chronic intestinal disease without a previous analysis of the urine; they require no emphasis here. In this article I shall consider the influence of the intestinal affections upon the urine and the alterations in its quality to which pathological conditions of the intestine give rise.

Amount.—Whenever a large quantity of fluid is lost through the intestinal tract, there is a diminution in the amount of the urine excreted. Thus we have oliguria in acute gastro-enteritis, dysentery, cholera morbus, and Asiatic cholera. In the last-named disease there is often total suppression. Medicaments causing a large watery stool cause a corresponding diminution in the amount of urine. The urine being concentrated, is highly colored and has a high specific gravity.

Albuminuria.—The occurrence of albumin in the urine as the result of choleriform diarrhea was pointed out in 1869 by Kjelberg (" Nordiskt. Med. Archiv," 1869). Singer and Koebler showed its presence in even mild cases of enteritis. The albumin occurs only in small quantity—often only a trace is present. It disappears, as a rule, with the subsidence of the acute symptoms. Its presence is probably due to a disturbance in the circulation (circulatory albuminuria). Fischl and Stiller attribute it to the sinking of the arterial pressure. Singer goes further, in that he holds it to be due to a disturbance in the nutrition of the kidneys due to the arterial ischemia. It is possible that a toxic influence as the result of the absorption of ptomains from the intestinal tract may be the cause in some cases (toxic albuminuria). (See J. C. Hemmeter, "On the Relation of Intestinal Putrefaction to Albuminuria," "Maryland Med. Jour.," July 24 and 31, and August 7, 1897; also B. Stiller, "Albuminurie bei Diarrhöe u. Obstipation," "Ungar. med. Presse," No. 3 u. 4, 1901.) In intestinal diseases associated with high fever, especially if prolonged, as in typhoid, albuminuria is of common occurrence—the febrile albuminuria, to which von Leyden called attention.

Cylindruria.—Casts are usually to be found accompanying the albumin. They are for the most part hyaline in character. At times they are quite abundant, while at others very few are to be found. Here and there a hyaline cast containing a few leukocytes or kidney epithelial cells or a partly granular cast is met with. In cases of acute enteritis and cholera nostras albuminuria and cylindruria may often be demonstrated. They have been found in cases of incarcerated hernia and intestinal stenosis. A patient suffering with incarcerated hernia under the care of Dr. McElfresh, at the University Hospital, showed most beautifully this form of cylindruria. On entering the hospital this patient's urine was fairly loaded with casts, most of which were granular and hyaline, although a number of epithelial casts were present. The urine also contained a considerable amount of albumin. This condition persisted for several days after the operation, and on leaving the hospital the urine still contained casts in large numbers, so that I thought the man was nephritic. An examination made about two months thereafter showed a normal urine, both the casts and albumin having entirely disappeared. Israel reports hemorrhagic nephritis in a case of volvulus of the sigmoid flexure ("Berlin. klin. Wochenschr.," Bd. L, 1892).

Albumoses and Peptones.—Pocanowski and Maixner showed that the various forms of ulceration of the intestines are attended by peptonuria. Part of the peptone that is formed from the digestion of food in the intestines is thought to be absorbed through the ulcers and to pass directly without change into the blood (enterogenous peptonuria). Chvostek and Stromeyer observed that the administration of a large amount of peptone in a case of tubercular ulceration was followed by the appearance of peptone in the urine. This observation seems to lend confirmation to the above-mentioned view. On the other hand, it is not improbable that the septic material at the base of the ulcers is absorbed and gives rise to the peptonuria (pyogenous peptonuria). Although albumin and peptone are often present in the urine of patients suffering with ulceration of the intestines, they are not invariably found, so that while the finding of these substances speaks for ulceration, their absence does not permit of any inference being drawn. For the methods of determining their presence see the chapter on Examination of the Feces.

Indicanuria.—Indican occurs normally in small quantities in the urine as the result of the oxidation of the indol and its combination with the sulphate of potassium, forming potassium indoxyl sulphate ($C_8H_6NKSO_4$). The chemical reaction is probably as follows:

$$\underset{\text{Indol.}}{C_8H_7N} + O = \underset{\text{Indoxyl.}}{C_8H_6(OH)N}$$

$$C_8H_6(OH)N + HKSO_4 = \underset{\text{Indican.}}{C_8H_6NKSO_4} + H_2O.$$

Indol is formed as the normal product of intestinal digestion, and has been shown by Radziejewsky, Kühne, and Nencki to be the result of bacterial fermentation of proteid food material. The investigations of Jaffé, Salkowski, Bowman, and Brieger have definitely established the relation of indican to indol. The amount of indican excreted under normal conditions varies directly with and almost proportionally to the amount of the proteid nourishment taken. The daily amount is from five to twenty milligrams. Its presence is best determined by Jaffé's test: To five cubic centimeters of urine an equal amount of concentrated hydrochloric acid is added. A concentrated solution of calcium or sodium hypochlorite is added drop by drop until the mixture becomes no darker. After each addition of the hypochlorite the tube is thoroughly shaken. By this method the indican is oxidized to indigo-blue. Chloroform (three cubic centimeters) is then added, which takes up the indigo-blue. An excess of hypochlorite solution must be avoided, as the indigo becomes thereby further oxidized to a colorless product.

It is well to perform the test several times with varying amounts of hypochlorite until a maximum of the blue color has been found. This occurs when all the indican has been converted into indigo-blue and none has been decolorized by overoxidation. For the quantitative estimation Salkowski's method is to be recommended: It is first ascertained how much hypochlorite is required to bring out the indigo-blue most intensely in a given quantity of urine, as already described. If the urine is rich in indican, 2.5 c.c. or 5 c.c. are used, otherwise 10 c.c. are used. This is then treated with an equal amount of concentrated HCl, and the necessary amount of hypochlorite, as ascertained in the preliminary test, is added. The mixture is neutralized with sodium hydroxid and rendered alkaline with sodium carbonate. The indigo-blue is collected upon a filter and the filter is then washed with water until the residue is no longer alkaline. It is dried and repeatedly extracted with chloroform, which dissolves out the indigo-blue. This chloroform extract is next diluted with chloroform to an even number of centimeters, and poured in a glass vessel with parallel sides. It is now compared with a known dilution of indigo-blue placed in a similar vessel. This is thereupon diluted until both solutions are of the same shade. From the amount of the dilution necessary, the amount of indigo-blue in the urine is easily determined. Jaffé has found the amount to reach, under pathological conditions, as high as 150 mg. in the twenty-four hours. For clinical purposes the quantitative method is not necessary. By using definite quantities of urine (5 c.c.), hydrochloric acid (5 c.c.), and chloroform (3 c.c.) in every case examined, one learns by a little experience the shade obtained from normal urine, and can not only readily recognize when it is increased, but also gain an approximate idea of the amount of the increase. Ortweiller has formulated the three conditions for an increased excretion:

1. There must be sufficient proteid material in the small intestine, which shall undergo bacterial digestion.

2. There must be sufficient time given for bacterial action to occur.

3. The mucous membrane must be in a condition to absorb the bacterial products.

From these statements it will be seen that in those conditions which are accompanied by impaired motility of the intestines and increased vulnerability of its mucous membrane an increased amount of indican will be formed. In occlusion of the *small* intestine, as was first shown by Jaffé and confirmed by Senator, Leube, von Jaksch, and Ortweiller, an enormous amount of indican is formed—ten to fifteen times the normal amount. In experiments on dogs Jaffé found the increase to occur during the first twenty-four hours after the ligature was applied; but the greater increase occurred on the second or the third day. Nothnagel's experience is also that the marked increase does not occur until after the first twenty-four hours. In occlusion of the *large* intestine Jaffé found, in his experiments on dogs, no increase or an insignificant increase as compared with that attending occlusion of the small intestine. Clinically it has been demonstrated that marked indicanuria can occur in cases of occlusion of the large intestine, but only after a number of days after a secondary stagnation in the small intestine has developed. If, then, in cases of intestinal occlusion, indican is present in large amounts in the first two or three days, there being no other cause for indicanuria, it speaks for involvement of the small intestine; the absence of indican, for occlusion of the colon. After a duration longer than three days the absence of indicanuria speaks for colon disease, but its presence permits no inference to be drawn. In diffuse peritonitis an enormous increase may occur, especially in the acute form. The peritoneal inflammation causes a paralysis of the muscular coats of the small intestine, with a consequent retention and putrefaction of the contents. The presence of pus in the peritoneal cavity also gives rise to indicanuria. In chronic constipation due to atony of the small intestine there is *no* increased indicanuria. I have examined the urine of patients who have not had a fecal evacuation for from three to ten days, and have never been able to demonstrate a marked increase. Brieger and Ortweiller have produced constipation by the administration of opium, and found the urine poor in indican. Increased indicanuria has been found to occur in a number of conditions associated with diarrhea, but when there is an involvement of the mucous membrane. It occurs in acute gastro-enteritis, typhoid fever, tubercular ulceration, cholera nostras, and Asiatic cholera. The explanation is to be found in the third condition of Ortweiller—the increased vulnerability of the mucous membrane. In these conditions, although the intestinal peristalsis is increased, yet there is an increased bacterial activity with abnormal putrefaction, and the condition of the mucosa allows the ready absorption of the products. In diarrhea as the result of giving purgatives and in the cases of nervous diarrhea I have examined there was found rather a diminution of the amount of the indican. In interpreting the diagnostic significance of indicanuria, the other nonintestinal sources of its production must be kept in mind and excluded. Its source may exist in putrefactive processes in other tracts and cavities. Von Jaksch has found it in cases of empyema, gangrene of the lungs, and putrid bronchitis. Its presence in purulent peritonitis has been mentioned. It has been found in diseases of the stomach associated with subacidity and anacidity; also in gastric carcinoma and stenosis of the pylorus due to any cause.

Indirubin is often found accompanying indican. This substance was found by Rosenbach to yield indigo-red on boiling the urine and adding concentrated nitric acid drop by drop. The formation of a deep red color is due to the presence of indigo-red. It has the same significance as indican.

Ethereal Sulphates.—Besides the combination of indoxyl with sulphates, other ethereal compounds are formed and appear in the urine as sulphids. They are the sulphates of phenol, kresol, skatoxyl, and pyrocatechin or orthodihydroxy-benzene. These substances were discovered and studied by Baumann ("Pflüger's Archiv," Bd. XII u. XIII). The daily amount excreted normally varies between the wide limits of 0.094 gm. and 0.62 gm. (see von den Velden, "Virchow's Archiv," Bd. LXX). The proportion of the ethereal sulphates to the preformed sulphates in the urine is on the average 1 to 10, but the variations from this proportion under perfectly normal conditions are often so great that to judge of the abnormal deviations by it is not justifiable. Great importance has been laid upon this ratio for judging the intensity of intestinal putrefaction; but Fr. Müller, Salkowski, and von Noorden have shown that the ratio is of minor importance. Of much more value is the absolute amount of the ethereal sulphates.

Baumann and Morax ("Zeitschr. f. physiol. Chem.," Bd. x, 1886) showed that cleansing the intestinal canal of a dog by calomel causes the ethereal sulphates to disappear. On the other hand, if intestinal putrefaction is increased by neutralizing the hydrochloric acid of the stomach with carbonates the amount of ethereal sulphates is abnormally increased. By the estimation of these compounds, then, we gain an insight into the degree of the putrefaction. If one desires to disinfect the bowels previous to the operation of resection, the disappearance of the ethereal sulphates is an indication that the disinfection has been partially accomplished (see Kast and Boas, "Münch. med. Wochenschr.," No. 4, 1888). Absolute disinfection of the intestine by therapeutic means is impossible (see chapter on Intestinal Bacteria). The normal amount varies between such wide limits (see above), and the methods require so much time that, for ordinary clinical purposes, one is usually satisfied with the determination of the increase of one of these sulphates—viz., indican. When the indican is increased, the other sulphates are usually increased; but, on the other hand, the total ethereal sulphates are increased in conditions when the indican is not, as in acute infectious diseases (diphtheria, erysipelas, etc.) (Brieger, "Zeitschr. f. klin. Med.," Bd. III, 1881).

Diazo Reaction.—Boas, in his work on "Diseases of the Intestines," has called attention to the occurrence of Ehrlich's diazo reaction in cases of tuberculosis of the bowels. He holds that this reaction is seldom missed in these cases. In a number of cases of this type examined by us the reaction was marked. It must be recalled, however, that this reaction is present in long-standing cases of pulmonary tuberculosis, in which there is liable to be an intestinal ulceration. Thus, Michaelis ("Berlin. klin. Wochenschr.," March 26, 1900) found that out of 167 cases of phthisis examined, the reaction was positive in 111 cases—*i. e.*, in over 66 per cent.

The reaction is therefore of use in diagnosing tubercular ulceration from other forms in which the reaction does not occur, as carcinomatous ulceration, but it is of no value in the diagnosis of ulceration *per se*. The Ehrlich diazo reaction is also a very delicate test for the presence of bilirubin in the urine; the test depends upon the formation of azobilirubin (F. Pröscher, "Centralbl. f. innere Med.," No. 7, 1901).

Acetone and diacetic acid have been frequently observed in the urine in cases where there was wasting, with consumption of the tissue albumins, due to any cause. In cases of intestinal disease accompanied by wasting acetonuria is of common occurrence. Whether there is an acetonuria due to abnormal disintegration of food albumins in the intestines is as yet a matter of doubt. In other words, is there a true enterogenous acetonuria or is its presence due to a disintegration of the albumins of the body—inanition acetonuria? H. Lorenz ("Zeitschr. f. klin. Med.," Bd. XIX, 1891) found acetone and often diacetic acid in all cases of gastro-enteritis examined. In general the amount of acetone was proportional to the intensity of the symptoms. He found it also in intestinal ulceration and occlusion; in intestinal disturbance due to teniæ; and in several cases of peritonitis accompanied by vomiting and diarrhea. In a number of these cases acetone is found in the stool and stomach-contents. The fact, further, that it has been found in the contents of a dilated stomach by von Jaksch, von Noorden, and Lorenz seems to be evidence of a genuine digestive acetonuria. It must be remembered that the acetone in the stomach-contents may be due to an excretion from the blood into the stomach analogous to the presence of urea in the contents in cases of deficient kidney functioning. Furthermore, in all the cases in which Lorenz has found acetonuria there was a marked disturbance of nutrition with consequent wasting. *Diacetic acid* is very rarely found without acetone. Its presence is of much more importance than that of acetone. It indicates a serious pathological process, and gives to the case an unfavorable prognosis. In diseases of children it is of less serious import, the case often terminating in recovery in spite of its presence.

Gerhardt's Bordeaux reaction, for detecting diacetic acid in the urine, is obtained by adding to the urine a few drops of a solution of iron chlorid diluted until it has the color of Rhine wine, to precipitate phosphates; it is then filtered and a few more drops are added. A Bordeaux-red color with a violet-red foam results, the color disappearing on the addition of sulphuric acid. The filtration is not absolutely necessary.

LITERATURE.

1. Kjelberg, "Nordiskt. Med. Archiv," Bd. I, 1869.
2. Singer, "Prager med. Wochenschr.," Bd. XII, S. 9, 1887.
3. Kobler, "Wien. klin. Wochenschr.," Bd. III, S. 531, 1890.
4. Fischl, "Prager Vierteljahresschr.," Bd. CXXXIX, S. 27, 1878.
5. Schiller, "Wien. med. Wochenschr.," Bd. LXXVIII u. LXXIX, 1890.
6. Leyden, "Zeitschr. f. klin. Med.," Bd. III, S. 161, 1881.
7. English, "Oesterr. Jahrbücher," No. 2 u. 3, 1884.
8. Von Engel, "Prager med. Wochenschr.," No. 14, 1894.
9. Pocanowski, "Zeitschr. f. klin. Med.," Bd. IX, 429, 1885.
10. Maixner, "Zeitschr. f. klin. Med.," Bd. VIII, 234, 1884.
11. Chvostek und Stromeyer, "Wien. klin. Wochenschr.," No. 47, 1896.
12. Radziejewsky, "Du Bois' Archiv," Bd. XXXVII, 1870.
13. Kühne, "Bericht d. deutsch. chem. Gesell.," Bd. VIII, 1875.
14. Jaffé, "Centralbl. f. d. med. Wissensch.," Bd. X, 1872.
15. Salkowski, "Bericht d. deutsch. chem. Gesell.," Bd. IX, 1876.
16. Baumann, "Pflüger's Archiv," Bd. XIII, 1876.
17. Baumann und Brieger, "Zeitschr. f. physiol. Chem.," Bd. III, 1889.
18. Salkowski, "Virchow's Archiv," Bd. LXVIII, S. 407, 1876.
19. Ortweiller, "Mittheil. aus der Würzburger Klinik," Bd. II, S. 153, 1886
20. Krokiewicz, "Wien. klin. Wochenschr.," No. 29, 1898.
21. Fr. Müller, "Zeitschr. f. klin Med.," Bd. XII, S. 63.
22. Von Noorden, "Zeitschr. f. klin. Med.," Bd. XVII, S. 529.
23. Salkowski, "Zeitschr. f. physiol. Chem.," Bd. XII.
24. Schmidt, Adolph, "Untersuch. der menschl. Feces."

CHAPTER X.

DIAGNOSTIC SIGNIFICANCE OF CHEMICAL AND PHYSICAL EXAMINATION OF THE GASTRIC FUNCTIONS FOR INTESTINAL DISEASES.

A knowledge of the peristaltic and secretory function of the stomach, as well as of its chemistry, is of far-reaching importance in the diagnosis of intestinal diseases. Besides being of great therapeutic interest to know whether the stomach is implicated in the various intestinal diseases, abnormal states of the stomach in themselves are frequently the direct cause of the intestinal abnormality, which has come to the foreground and completely submerged the original gastric disturbance.

Gastritis.—Acute and chronic catarrh of the stomach is often the forerunner of enteritis, particularly duodenitis. The author has pointed out previously (Hemmeter, "Icterus and Cholangitis," "Bulletin of the Maryland University Hospital," March, 1898) that two-thirds of all cases of simple catarrhal duodenitis associated with icterus present the signs of hypo- or achylia gastrica, and that the symptoms and course of this form of icterus were, in his experience, markedly benefited by the administration of HCl. This, of course, has no reference to those cases of icterus due to cholelithiasis. The fermentative masses which replace the normal gastric chyme in acute gastritis are prominent causes of duodenitis.

Ulcer.—Leube ("Spec. Diag. d. inneren Krankheiten," 1898, Bd. 1, S. 290), in differentiating between gastric and duodenal ulcer, expresses the opinion that a low degree of HCl with the symptoms of ulcer speak for its location in the duodenum. One of his undoubted cases of duodenal ulcer had an amount of free HCl equal to 0.16 per cent. The relation of gastric hyperacidity to peptic ulcer has been set forth in the author's book on "Diseases of the Stomach," second edition, page 501. Suffice it to say that the hyperchlorhydria which Riegel first emphasized as being constantly associated with gastric ulcer was later confirmed in many analyses by von den Velden, Jaworski, Ewald, Boas, and myself, so that it is a valuable clinical sign as far as the gastric ulcer is concerned. In order to establish a low amount of free HCl or absence thereof as a diagnostic sign of duodenal ulcer, when the symptoms suggest this disease, a much larger number of clinical observations than are reported at present is required.

Neoplasms—Carcinoma.—The gastric secretion and peristalsis, as a rule, continue normal in intestinal carcinoma to the very last stages of the disease, provided the neoplasm is not located in the duodenum or upper part of the jejunum. Neoplasms of this region present characteristic symptoms which are not observed in those located in other parts of the bowel. These conditions have been treated in a separate chapter. It is very helpful to be able to exclude gastric carcinoma in abdominal tumors of doubtful location. In a case of supposed tumor of the stomach, palpable about the middle of the larger curvature, I was able to exclude gastric carcinoma from the normal amount of HCl, the absence of lactic acid, and the normal peristalsis; and at the operation the tumor was found in the transverse colon, but was fixed to the greater curvature by adhesion, so that on distention and palpation it gave every evidence of being a gastric neoplasm. The analysis of gastric contents may be helpful in this way in distinguishing neoplasms of the pancreas, liver, and omentum from those of the stomach itself. While a normal gastric chemistry constitutes an argument against the existence of gastric carcinoma, an abnormal gastric chemistry does not speak against the existence of an intestinal affection. In fact, in duodenal neoplasms, as we shall see later, the gastric chemistry is, as a rule, abnormal, especially in the later periods of the disease. An interesting case is reported by Pawlaski ("Berlin. klin. Wochenschr.," 1892, No. 12), in which the symptomatology was suggestive of a gastric neoplasm, associated with advanced dilation of the stomach, but the autopsy showed a primary carcinoma of the cecum. The gastric tumor was present, but it was caused by secondarily carcinomatous lymph-glands which had compressed the pylorus.

Duodenal Stenoses.—In the chapter on Enterostenoses I have described the various types of stenoses of the duodenum. To repeat in brief what is said there: There are two main forms, classified according to the location—(a) those above and (b) those below the papilla, which mark the entrance point of the common gall- and pancreatic duct. The suprapapillary stenoses—those located between the papilla and the pylorus—can not be diagnosed by analysis of gastric contents, because the obstruction prevents the entrance of duodenal contents into the stomach. But the infrapapillary stenoses—those which prevent the duodenal secretion from reaching the jejunum—are marked by the constant presence of considerable bile in the stomach when the contents are taken in the morning before breakfast. Boas was the first to point out that under such conditions pancreatic juice, and probably also a small amount of succus entericus, enters the stomach together with the bile, and that the gastric secretion may be replaced by a duodenal digestion, especially if the amount of HCl secreted is low. If an abundance of HCl is still secreted, no marked change in the character of the digestion occurs. The digestive processes in the duodenum, the preparatory accumulation of duodenal secretion, and some of the digestive properties of the combined mixture of gastric chyme and duodenal secretions—see the experiments of Chittenden and Rachford (*l. c.*) on this subject—have been stated in a separate chapter. Our knowledge of the nature of duodenal digestion is still problematic in many respects. Many of the assertions commonly found in text-books on physiology concerning intestinal indigestion are pure assumption, and by no means controlled by experiment or histological investigation. We must be prepared to see many favorite interpretations concerning intestinal indigestion and autointoxication modified in the future. Lukjanow expresses the hope that ("Die Pathologie der Verdauung," Leipzig, 1899, p. 231) by my method of duodenal intubation new light may be thrown upon this subject. Boas ("Darmkrankheiten," *l. c.*, p. 136) suggests that the presence of pancreatic ferment in the stomach with a palpable tumor in the region of the descending arm of the duodenum speaks for pancreatic tumor, and the absence of the ferments is an argument against pancreatic tumor. I have had no experience with the presence of pancreatic enzymes occurring simultaneously with a palpable tumor in that region, but so far as the absence of pancreatic enzymes in the gastric contents being a contraindication of pancreatic tumor is concerned, I recall two cases in my experience where pancreatic tumor was found at operation, but there were no pancreatic enzymes in the gastric contents at analyses taken on four different occasions in each case. I, therefore, do not look upon this as a reliable sign, because the pancreatic neoplasm may be present and stenose the pancreatic duct, thus preventing the outflow of pancreatic juice.

Jejunal Stenoses—Relation to Gastric Contents.—The gastric contents in jejunal stenosis resemble the jejunal chyme—*i. e.*, they are slightly fecal, of a yellowish-brown color, and do not present at any time the reactions of pure bile and pancreatic juice. The further particulars concerning this phenomena have been discussed in the chapter on Enterostenoses.

The Gastric Secretions and Intestinal Complications.—Achylia gastrica is frequently associated with diarrheas, and in cases of chronic diarrhea it is very important to analyze the stomach-contents. Many such cases in my own practice have been relieved by the simple administration of HCl (Hemmeter, "Diseases of the Stomach," second edition, p. 328; also "The Logic of HCl Therapy, etc.," in "American Medicine," Nos. 3 and 4, April 20 and 27, 1901). Boas (*l. c.*, p. 137) also admits of a certain connection between anacidity and chronic diarrheas, and Oppler ("Deutsch. med. Wochenschr.," 1896, No. 32) has written a very scholarly article on the complete suppression of gastric secretion in chronic diarrheas.

Reversely, hyperacidity is frequently observed in causative relation with chronic obstipation, so that a relief of the hyperchlorhydria by alkalis cures the constipation. Sometimes this symptom can be relieved by morphin, opium, or belladonna in these cases, because these drugs inhibit the secretion of HCl. Analysis of gastric contents is then as important in the course of diarrhea as it is in the course of constipation.

Gastrectasia and Gastroptosis.—Both dilation as well as displacement of the stomach may be associated with deranged gastric chemistry, which in turn is of influence in the pathogenesis of intestinal diseases. In some forms of these diseases medication by the mouth, intended for the relief of intestinal symptoms, reaches the intestine either very late or not at all, because of the existing gastric motor insufficiency. Gastroptosis and gastrectasia may also simulate displacements of the colon, and for these reasons alone a study of the contents of the stomach and its size and location is indispensable when we are treating obstinate intestinal diseases.

CHAPTER XI.

DUODENAL INTUBATION.

f the methods nowadays employed in the physical diag-
ases of the intestine have gained a hold upon the confi-
icians which is comparable only to the reputation of aus-
d percussion and examination of the sputum in pulmonary
d examination of the urine in diseases of the kidneys.
ds are the microscopic examination of the feces, the ex-
r test lavage of the colon, direct inspection of the rectum
d, and examination of curettings gained during this direct

The method to be described at present can not claim
tation for being productive of diagnostic conclusions;
s it is so far the only method from which any hope may
or further and future knowledge concerning the physio-
athological chemistry and permeability of the duodenum
n, a description of this method appears justifiable. Intuba-
duodenum may give valuable information in two ways:
form us concerning the permeability of the pylorus and
nd jejunum, and (2) we may, under favorable conditions,
spirate the duodenal chyme and subsequently analyze it
the gastric chyme is drawn and analyzed for purposes of
Lukjanow (" Pathol. d. Verdauung," p. 231) emphasizes
ity of further knowledge concerning the chemical pro-
e beginning of the small intestine, and hopes for the
f a technic which may be serviceable for obtaining and
the intestinal contents of patients as drawn from the

he only way in which such knowledge could be
o collect the discharge from fistulæ of the small in-
alyze them. But few fistulæ have occurred in the
have referr he celebrated case of Busch in the
amination ge from

such fistulæ has given valuable information, the chief objection to them is that such patients can not be considered as being in a normal condition—an intestinal wound discharging upon the abdomen constitutes a serious disturbance of normal functions.

Anticipating the great needs of diagnosis by increased knowledge of the chemical processes in the duodenum, Boas attempted to force the contents of the beginning of the intestine back into the stomach through massage, and thence to draw it by means of the lavage tube ("Zeitschr. f. klin. Med.," Bd. XVII, H. 1 und 2, 1890, S. 155).

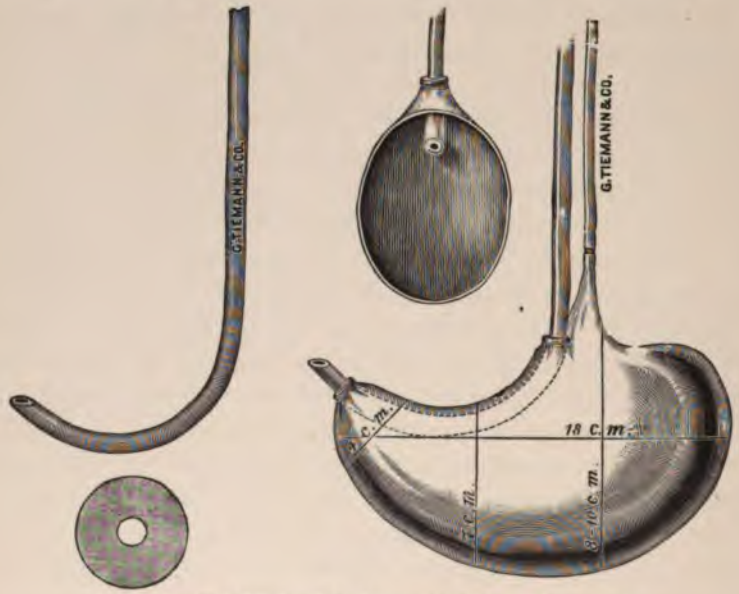


FIG. 15.—APPARATUS FOR OBTAINING INTESTINAL CONTENTS.

The first methodical procedure to obtain the contents of the duodenum in the human subject was described by the author in 1896 (J. C. Hemmeter, "Intubation of Duodenum," "Bulletin of Johns Hopkins Hospital," vol. VII, No. 61), and later in Boas' "Arch. f. Verdauungs-Krankheiten," "Versuche ü. Intubation des Duodenum," Bd. II, S. 85. The following is an abstract original report describing the author's method:

This method consists, in the first place, of the introduction of a thin elastic rubber bag into the stomach. This bag, when folded over a tube which runs through it, does not occupy so much space as an ordinary stomach-tube, and has the exact shape of the human stomach when it is distended by blowing it up within that organ, to which it fits itself exactly, and is closely applied to the gastric walls.

The intragastric bag is distended by the pressure apparatus shown in figure 16. The graduated bottle *A* is full of water and elevated above the bottle *B*, which is empty and also graduated.

The stomach-shaped bag, *C*, when it reaches the stomach, is connected with the lower empty bottle, *B*. Then the stop-cock connecting *A* with *B* is opened, and the water runs from *A* into *B*, displacing the air in *B*, which distends the bag *C* within the stomach, filling it entirely. As is observable on this bag, a guide is contained in it, running along the dotted line parallel to the lesser curvature. In this guide the duodenal tube is inserted, lubricated with glycerin before the bag is pushed into the stomach. This tube is provided with very thick walls, by virtue of which it is not easily kinked or bent upon itself (see cross-section, Fig. 17).

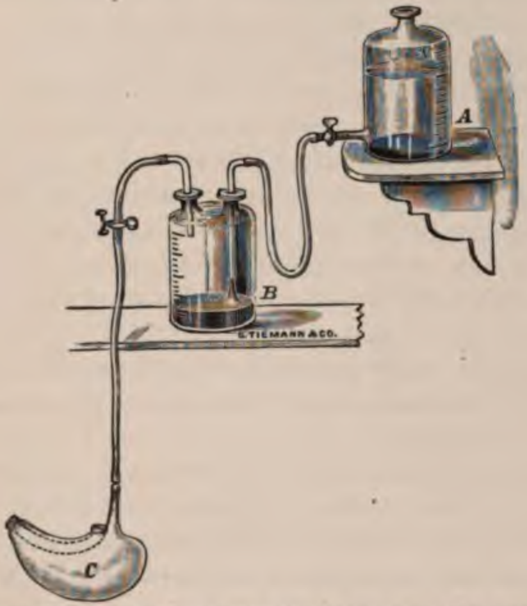


FIG. 16.—PRESSURE BOTTLES FOR DISTENDING THE INTRAGASTRIC BAG DURING DUODENAL INTUBATION.

The relation of the thickness of the walls to the diameter of the lumen is shown in the cross-section of figure 15. When the intragastric bag is inflated, it fills the stomach entirely. The duodenal tube lies in its sheath or guide, and, on being pushed onward from the mouth, it is not possible for it to go anywhere else except through the pylorus into the duodenum. In the illustrations it can be seen that the bag is not distended by the duodenal tube, but a separate very small tube runs down the esophagus, ending in the bag, serving the purpose of its distention. Both tubes together do not occupy so much space as an ordinary stomach-tube.

By this method we can obtain the contents of the intestine, and the gut may in any of its parts be reached with safety.

After known test-meals it is possible, after they have passed from the stomach into the duodenum, to draw out samples from this part and subject them to analysis. By alternately distending any part with air or water we will be enabled to locate the part by the percussion sound on the outside of the abdomen, and the distance it is located from the mouth can be seen from the length of tube introduced.

Small electric lamps may be introduced into the duodenum as they are into the stomach, and the location and condition recognized by electrodiaphany.

Hitherto, in all experiments on this subject, it has been impossible to obtain either the pancreatic or biliary secretion in a pure condition; this is due to the fact that both the pancreatic and the common gall-duct empty into the descending portion of the duodenum very near each other.

In May, 1897, I had under observation a female patient who had suffered repeated biliary colic. At times she passed small stones without giving her much pain—at least they were found in the stools without having given her any colic. She was deeply jaundiced and willing to undergo an operation to be relieved. Through the comparatively thin abdominal walls we were able to feel numerous stones in the gall-bladder. She consented to an attempt at intubation of the duodenum to determine whether there was any bile secreted. The duodenum was entered without difficulty, and cleansed by running in and aspirating out distilled warm water. Twelve hours afterward, no food having been taken in the mean while, the duodenum was again intubated according to our method, and washed with 100 c.c. of warm distilled water.

On being aspirated, the water was still clear, but viscid and sticky, similar to a solution of egg-albumen. It contained no bile-pigments nor cholesterin, and was free from taurocholates and glycocholates. It was colorless and odorless, and seemed very rich in some form of albumin. That it was a solution of pancreatic juice was proved by its digesting fibrin and serum albumin.

The juice obtained in this manner digested from 85 to 95 per cent. of Merck's dried serum albumin in the digestorium at 100° F. in two hours. The amylolytic and fat-decomposing property of the juice was determined in a similar manner. One is therefore justified in concluding that in this case the pancreatic juice was obtained almost pure, as there were no bile elements contained in it, the bile being prevented from entering the duodenum by a calculus or catarrhal occlusion in the common duct. This is an evidence that these are cases of occlusion or stenosis of the common gall-duct not involving the pancreatic duct. As there are also pancreatic calculi, or occlusions of the duct by neoplasm or catarrhal swelling, it is conceivable that we may yet be able to obtain the bile in a pure condition, and free from pancreatic juice, from the human subject, without operation.

The secretion of Brunner's and Lieberkühn's glands will, however, always constitute an admixture of these juices.

The second method of duodenal intubation was invented by Dr. Franz Kuhn, of Riegel's school, and published in the "Münch. med. Wochenschr.," No. 29, July, 1896, S. 674. Further communications appeared in the "Arch. f. Verdauungs-Krankheiten," Bd. III, S. 19 ("Sondierungen am Magen, Pylorus, und Dünndarm des Menschen"). Kuhn's method is more practical than my own, because the instruments are simpler in construction and permit of entrance into the duodenum with greater surety than the method by means of the intragastric balloon sheath. It consists essentially of a tube built up of four different parts, one ensheathed in the other. While this gives the tube sufficient rigidity to penetrate the pylorus, it is not stiff enough to do harm in the hands of a person who has learned the method from a capable teacher. I should remark here that intubation of the duodenum had always best be learned from a capable master, for, like gastroscopy, it can hardly be acquired by self-teaching.

The three parts of the Kuhn intubation tube are seen in figure 17, page 269, in cross-section. They consist of: (1) A rubber tube or covering for the whole instrument, the walls of which increase in thickness toward the lower end of the tube (see *g*, Fig. 17, p. 269); (2) inside of this is a very flexible metallic tube, which is constructed by spirally winding a metal band of copper bronze around a solid steel rod; the band of copper bronze has an S shape on cross-section; it can be seen at *sch*, in parts 1 and 3 of figure 17, page 269; in winding this band the grooves of the S fit into each other so snugly that the metal spiral tube will hold water when it is perfectly constructed. At the same time it is capable of undergoing serpentine undulations and bendings, and of exhibiting a marvelous flexibility. (3) Within the metal spiral tube there runs a smoothly polished spiral, constructed of relatively thick steel wire, wound tightly, and making a very elastic rod with considerable springiness. This spiral tube moves easily within the metal elastic tube; its lower end terminates in a little knob. The spiral hose described under (2), immediately adjacent to the rubber tube, would not have sufficient rigidity and springiness but for the third smoothly polished and tightly wound spiral. By pressure upon the innermost rod at the external part, which projects from the end of the instrument, the little knob at the duodenal end is pushed against the spiral tube described under (2), which rests in a ridge molded into the rubber tube and imparts to it curiously seeking and tactile motions. Such slight pressure on the external end of the apparatus, when the tubes are pushed into each other, when executed outside of the body, can, by skilful manipulation, give to the movements of the instrument the appearance of a living snake seeking its way over obstacles. (4) The fourth component of the Kuhn sound is a simple slender steel rod or wire which still further strengthens the rigidity of the whole instrument.

The various parts of the instrument are so constructed as to move one within the other with the greatest facility and without friction. In attempting intubation with the Kuhn instrument, it is important to establish the size and location of the stomach beforehand. Above all things it is necessary to assure one's self of the location of the pylorus. Kuhn has devised a special instrument—the balloon sound, figure 18, page 270—for this purpose. When distended within the stomach, this balloon can be palpated, in favorable cases, through the abdomen. He has also devised a simple esophageal sound (Fig. 19, p. 270) for determining the length and possible strictures in the esophagus. The metal parts of none of the sounds reach to the tip of the rubber sheath or incasing, thus insuring the necessary softness and elasticity at the duodenal end.

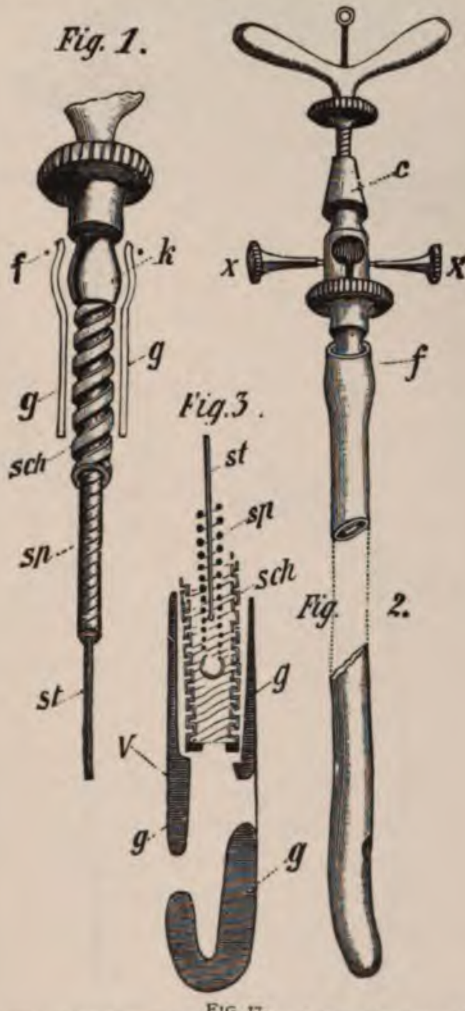


FIG. 17.

Fig. 1, diagram of cross-section of upper end of duodenal tube, showing outside rubber tube (*g*), the spiral metallic hose (*sch*), the second hollow spiral (*sp*), and the innermost steel guide (*st*).
Fig. 2, shows the upper and lower ends of the instrument incased in its rubber sheath.
Fig. 3, shows the lower end of the duodenal tube, with the gradual thickening of the rubber of outside tube, and illustrating the rubber shelf or ridge preventing the metallic spirals from reaching to the tip of the instrument.

Method of Introduction.—The Kuhn tube is introduced into the stomach like an ordinary stomach-tube; a previous knowledge of the exact size and location of this organ permits the operator to judge the location of the tube while it is in the stomach. I prefer to turn the patient on the right side after the tube has reached the stomach, and, if the abdomen is relaxed, the

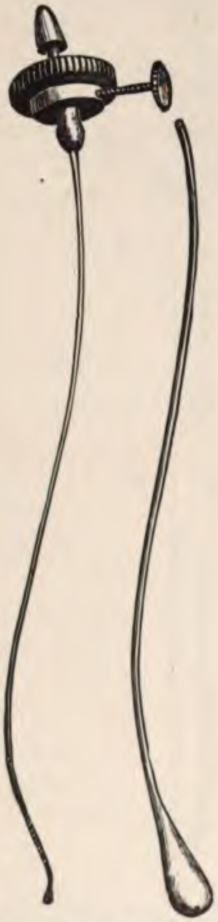


FIG. 18.—BALLOON SOUND OF KUHN.

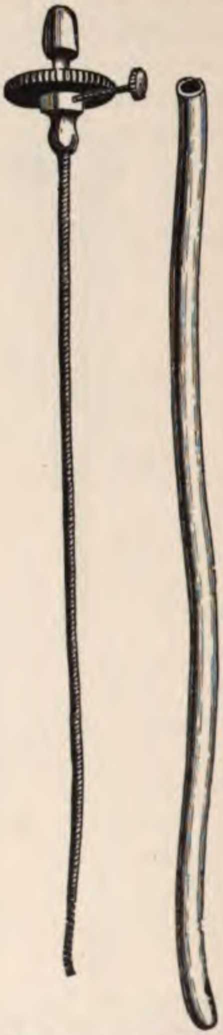


FIG. 19.—ESOPHAGEAL SOUND.

sound can be grasped with the fingers. The stomach, of course, must be previously emptied by lavage—this is not only necessary to facilitate the passage of the sound, but also to assure one's self that whatever is aspirated could not possibly come from the stomach, but must come from the duodenum. Figures 20 and 21 are schemata giving an idea of the manner in which the Kuhn tube glides along the stomach's greater curvature, as observed in cadavers and animals. Figure 20 shows what usually happens to ordinary rubber stomach-tubes when such an intubation is attempted with them. Even without massage and guiding the tube with the fingers from the outside intubation of the duodenum is possible by the Kuhn method. I have used it on thirty-four different patients, mainly for the purpose of assuring myself of the permeability of the pylorus. The chemical analysis of duodenal contents, which it is frequently possible to draw by this method, has not been undertaken in a sufficiently large number of cases to permit of any deductions valuable for diagnosis, but the following preliminary report will show the possibilities of the method.* In one patient in whom I

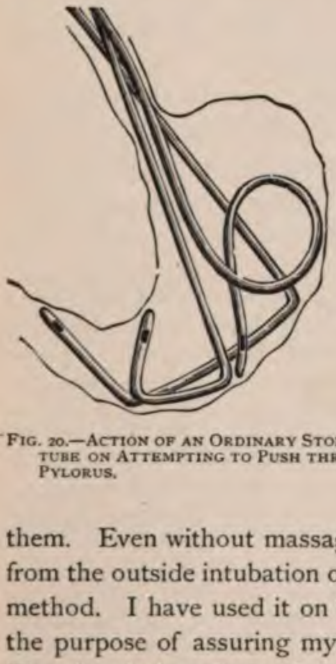


FIG. 20.—ACTION OF AN ORDINARY STOMACH-TUBE ON ATTEMPTING TO PUSH THROUGH PYLORUS.

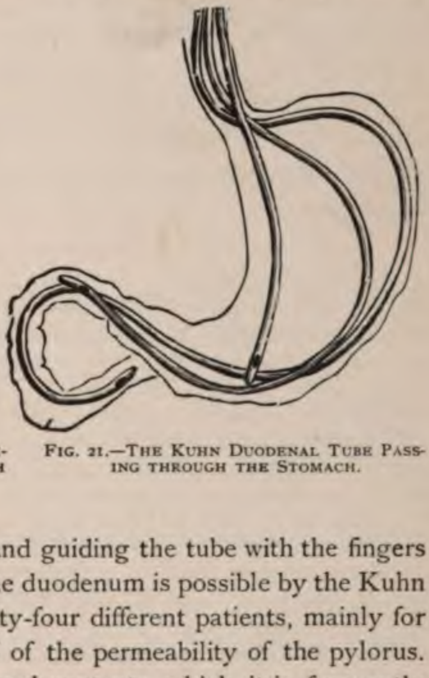


FIG. 21.—THE KUHN DUODENAL TUBE PASSING THROUGH THE STOMACH.

---

* The amylolytic, proteolytic, and adipolytic power of the duodenal chyme was tested by standardized solutions of pure starch, weighed amounts of fibrin, prepared according to the directions of Chittenden ("Amer. Jour. of the Med. Sci.," vol. III, p. 39), and weighed amounts of pure sterilized olive oil. It was found: (1) That these amylolytic

had intubated the duodenum successfully on five different occasions I had an opportunity of seeing the stomach at an operation undertaken for the relief of pyloric stenosis due to hyperplasia. The stomach was in no way injured; neither have I been able to discover, on the stomach of dogs, that the sound can produce any injury to the gastric mucosa when it is properly handled—and it should not be handled at all unless it can be properly handled. It should first be learned upon the cadaver and on animals before it is attempted on the living subject. One of the earliest references to duodenal intubation in American literature was published by Dr. Fenton B. Turck, of Chicago, in "Amer. Medico-Surgical Bulletin," July, 1895, vol. VIII, No. 13, page 772.

Our only hope for advance in diagnosis lies in such methods as gastroscopy, duodenal intubation, and sigmoidoscopy, which bring the interior of the stomach and intestines closer to our methods of investigation.

---

powers of the pancreatic juice as obtained from the duodenal chyme, mixed with bile varied under normal conditions in the same individual; (2) that they varied with different food substances; (3) that fat (olive oil) was a powerful dietetic stimulant to the secretion of pancreatic juice (J. P. Pawlow, "Arbeit der Verdauungsdrüsen," S. 160); (4) that the digestive activity of the various pancreatic ferments is more energetic when the gastric secretion and peristalsis are normal—in cases of achylia gastrica, where the secretion of HCl and of the gastric ferments is wanting, the digestive power of the pancreatic ferments was decidedly inferior to that from cases with normal gastric digestion. I examined four cases of achylia gastrica particularly for this point, and in all four the duodenal digestion was evidently inferior to that found in individuals with normal stomachs. It seems, therefore, that a normal secretion of pancreatic juice is dependent upon a normal secretion of HCl in the stomach. A disease of the stomach suppressing the secretion of HCl robs the pancreas of one of its chief stimulants to secretion. This is also in accordance with the observations of J. P. Pawlow (*l. c.*). Inasmuch as the HCl is a stimulant to pancreatic secretion, we may assume that anything which stimulates the secretion of HCl in the stomach will also enhance the digestive power of pancreatic juice. (5) It was found that the absence of bile from the duodenal contents—catarrhal icterus—reduces the power of the pancreatic ferments if they are still secreted; such a case was already described in the preceding, where the common gall-duct was stenosed by a small calculus. (6) There exists a compensatory arrangement whereby the alkalinity of the pancreatic juice increases as the acidity of the gastric juice increases, and, reversely, the alkalinity decreases as the acidity of the gastric juice decreases. These results have been mentioned merely to show the possibilities of this method and the large amount of work yet to be accomplished in this direction. The experiments are still in progress and will be published later.

PART SECOND.

DIET.—THERAPY AND MATERIA MEDICA OF INTESTINAL DISEASES.

CHAPTER XII.

PRINCIPLES OF DIETETIC TREATMENT.

*The Influence of Various Food Classes on Metabolism.—Caloric Requirements.—Isodynamic Relation of Proteids, Fats, and Carbohydrates.—Differences as Producers of Energy.**

Our knowledge of the subjects may be classified into two parts:

1. The various articles of food, their chemical and physical properties, and the methods of their preparation and combination.

2. The reaction of the organism to the introduced food in a normal and abnormal condition. This means, first, a description of the nutritive substances, and, second, the manner in which the organism responds to the food. The material which furnishes our nourishment comes from the animal and the vegetable kingdom; only two substances come from inanimate nature—these are water and sodium chlorid. An experience of thousands of years, handed from generation to generation, has made us familiar with a large number of animal and vegetable constituents that are serviceable for nutritive purposes. We call them food substances; they

---

* In writing this chapter the author has drawn largely from the following works: The research and clinical work by various investigators, and edited by E. von Leyden, "Ernährungs-Therapie u. Diätetik"; Munk u. Uffelmann (edited by C. A. Ewald), "Ernährung," etc.; F. Moritz, "Grundzüge d. Krankenernährung"; Th. Rosenheim, "Allgem. Diätotherapie" in "Lehrbuch d. allg. Therapie," von Eulenburg u. Samuel; Gilman Thompson, "Dietetics"; G. Linossier, "L'hygiène du dyspeptique," and the excellent dietetic research work by W. O. Atwater and associates, published by the l States Department of Agriculture.

are adapted, by their chemical and physical constitution, to be taken up in our organism and to be utilized in its metabolism; they are suitable to our taste; and they have, for the most part, no harmful side-effects.

By **metabolism** is meant the power that organized bodies possess of continually using up and renewing the materials of which they are composed. There are two kinds—viz., *anabolism*, which means the building up—the process of forming living tissue from the nutrient material; and *catabolism*, the process of breaking down or using up tissue. Let us proceed to consider the nutritive value of food. The chief foods may be divided into six groups: (1) Water; (2) salts; (3) proteids; (4) starches; (5) sugars; (6) fats and oils. The proteid or albuminous substances contain an element which is absent from the carbohydrates and fats—this element is nitrogen; for that reason the proteids are spoken of as nitrogenous and the carbohydrates and fats as nonnitrogenous. These three groups are the most important when we have to consider the nutritive values of food. They bring substances into assimilation in our bodies which are indispensable, or they prevent or reduce the amount of breaking down of tissues. By assimilation is meant the transformation of food into such nutrient condition that it can be taken up to form an integral part of the organism. From the economic standpoint of dietetics the other food substances, such as water and salt, are not so important, because we can, as a general thing, obtain them outside of the food substances.

As a rule, the nutritive value of food substances is measured by their constituent amounts of proteids, fats, and carbohydrates. The animal organism, our body, is undergoing never-ceasing changes. All substances of which we are composed undergo an uninterrupted alteration, exchange, and decomposition. The tissues of our bodies undergo extensive chemical transformation, the end-products of which are water, CO_2, and urea. These substances are given off in the liquid and gaseous excretions of the body. The waste of this breaking down is replaced from the food. The designation metabolism for this process is a very apt one; if the body maintains its weight, we speak of a balance of metabolism. All processes of life are dependent upon metabolism; they depend upon the fact that the potential chemical forces which are hidden in the organism become free—in other words, potential energy is converted into kinetic energy.

Potential energy means power or force which is not manifested, but latent, dormant, stored up: for example, the potential energy of a wound spring, of coal, or of a suspended body. Kinetic energy is power or force actually performing—it means evolved force. All foods contain potential energy, and it is converted into kinetic energy by the oxygen in the body. This is a process of burning up, or combustion. The oxidation of food within the body in the process of metabolism, to produce heat or force or to build up tissue, is a process of combustion, and it is generally accompanied by the formation of heat, though it also expresses itself by the mechanical work done by our muscles, the chemical and physical work done by the glands, and the mental work of the brain. The three main food substances—proteid, carbohydrates, and fats—are the fuel of the organism. When we undertake a chemical analysis of the body, we find much proteid or nitrogen substance, much of fat, but very little of carbohydrates. The proteid or albumin is an integral constituent of all cells; it is abundant in the muscles. The gelatinous substances form connective tissues and are contained in bones and cartilages. The fat is contained in all fatty tissues, particularly under the skin, in the peritoneal cavity, in the hollow bones, and in the muscles. The carbohydrates are present only in small quantities in the form of sugar in the blood and tissue juices, and by a starch-like substance called glycogen, found in the liver and muscles. Thus it comes about that a body which is in a state of temporary starvation—*i. e.*, when it must draw the requisites for its energy from its own substance—burns up only fats and proteids and no carbohydrates. But in the well-nourished body, which is supplied with an abundance of carbohydrates, their decomposition or burning up is also utilized to produce energy.

Water and mineral salts (NaCl), when they are utilized, undergo no combustion, but remain unchanged. As these materials are contained in all foods they can not be excreted until the organic foods of which they are a part are broken up. Water and salts, though indispensable to nutrition, yield no energy—only the proteids, carbohydrates, and fats can do this.

The chief uses of food are three: (1) Tissue building, to form the material of the body and repair its wastes; (2) heat-producing, to keep the body warm; (3) force-producing, to yield the muscular and other power for the work the body has to do.

Rubner found that in the organism in a state of absolute rest nearly the entire potential energy of the ingested food shows itself in the same physical (kinetic) form—namely, heat, when the organism must perform mechanical external work; of course, a certain amount of potential energy is consumed for this. Even in absolute rest a small amount of potential energy is consumed for the muscular work of the heart and lungs and digestive apparatus, but the amount of energy used for this force is insignificant in comparison to that turned out in the form of heat; during rest, excepting the muscular work just mentioned, practically all remaining energy is evolved in the form of heat. Rubner also proved experimentally that the different classes of food substances—proteids, carbohydrates, fats—may substitute one another according to definite rules and proportions.

The same amounts by weight of various materials used for fuel have a different heating value—a ton of wood will not give so much heat as a ton of coal; a ton of paper not so much heat as a ton of wood. The same is true of the foods: Fats have a much greater heat-producing effect than proteids or carbohydrates. Taking the same amounts by weight of these three food classes, the fats have a decidedly larger quantity of heat-producing energy than the proteids or carbohydrates. Heat and muscular power are forms of kinetic energy or force. The energy is developed from the potential energy of the food as it is consumed in the body. It can be measured in the laboratory by means of an instrument called the calorimeter. The unit commonly used is the calorie. A calorie is the amount of heat required to raise the temperature of one kilogram of water (about one quart) one degree Centigrade, or one pound of water four degrees Fahrenheit. Now,

1 gram of fat yields	9.4 calories.
1 " " carbohydrate yields	4.1 "
1 " " proteid "	4.1 "

This means that 1 gram of fat when oxidized will raise 9.4 liters of water 1° C.; but 1 gram of sugar will raise only 4.1 liters 1° C. From these figures we can easily calculate that 1 gram of fat $= \frac{9.4}{4.1} = 2.3$ grams of sugar. If one, therefore, feeds a dog on proteids or albumin and fat so that he just exactly keeps the balance of metabolism,—*i. e.*, that he adds no body material and loses none,—and if we wish to substitute sugar or any other carbohydrate in such a proportion that this metabolism balance shall be kept up, we must add or supply 2.3 grams of sugar for every 1 gram of fat we wish to replace. Only in this manner can the nitrogen balance or equilibrium be maintained ; if we substitute more or less of sugar for a given amount of fat, the balance is lost.

An analogous relation (isodynamic) exists between fat and proteid, fat and starch, fat and albumin.

In place of stating that 1 gram of fat and 2.3 grams of sugar can furnish the same amount of heat or calories, we express ourselves briefly by saying that they are *isodynamic* in proportion of 1 gram of fat to 2.3 grams of sugar.

For every kilogram or 2.2 pounds of body-weight a person requires :

During bed rest, ...	30–40	calories.
Out of bed, but not at work, ...	34–40	"
Moderate work, ...	40–45	"
Hard work, ...	45–60	"

By saying that the foods are isodynamic we mean that in definite proportions they produce an equal amount of heat when they undergo combustion after their digestion and assimilation in the body, and, as in the case which we illustrated by the fat to sugar, they can replace each other mutually with regard to their heat-producing properties. Similarly can they replace each other also with regard to their power of effecting muscular work and with regard to tissue building. When a body is insufficiently nourished, the amount of breakdown of albumin is at first not at all or only very slightly increased by physical work, but the consumption of fat is considerably increased thereby. The addition of a corresponding isodynamic amount of carbohydrates to the food will prevent the increase of this fat consumption. This shows that the nonnitrogenous foods—the fats and the carbohydrates—are the source of muscular power, because the organism, when it requires an increased amount of force or strength over and above the amount of energy that can be furnished by the ingested food, does not consume its own albumin or proteid, but only its fat.

When we say that the body, under those conditions, does not use its own albumin, we refer to the organized albumin which composes its protoplasm; we believe that the body can and does utilize the albumin contained in the food for muscular force. This non-organized albumin is capable of being transformed into carbohydrates and fat in the body; therefore it is not probable that there are distinct differences with regard to the rôle of the organic food substances as carriers of energy in producing heat or muscular force, because the body can obtain these energies from all three classes alike, though, under usual conditions, muscular force is derived from the fats and carbohydrates. Can the food substances mutually replace each other in tissue building or supplying the regular losses in albumin and fat which occur by the activity of the organs? Carbohydrates and fats can replace each other for this purpose, but proteid or albumin can not be replaced. The organism consists of preponderance of albumin and fat, and although carbohydrates are an integral part of its chemistry, because they occur in the blood and juices and liver, nevertheless they occur in much smaller quantities than the other two food elements. There are two sources for the percentage of carbohydrate in the body: (1) The carbohydrate in the food and (2) the albuminous or proteid bodies from which carbohydrate, glycogen, and grape-sugar can arise. Carbohydrates can not be formed from fat.

There are three sources for the fat in the body: (1) The fat of the food; (2) the carbohydrates; (3) the proteids. Both carbohydrates and proteids can, by synthetic processes, be transformed into fat. Fat is deposited most easily in the body when it is introduced as such; next it is formed from carbohydrates, but very large quantities of proteid must be introduced to effect a deposition of fat. Fat can not be broken down so readily as proteid, but proteid and carbohydrates are very easily broken down. The breaking down of proteid can be increased to almost any desired degree by the additions of corresponding amounts of proteid in the food. The organism does not store up fat made out of proteid until it can no longer break down the proteid which is introduced in a given time; it therefore requires a great deal of albumin or proteid in the food if we desire to make fat out of it to the exclusion of carbohydrates and fats in the diet.

The facility with which proteid is decomposed seems contradictory to the fact already emphasized, that the organism, in performing muscular work, does not consume its incorporated albumin or proteid, and also to the fact that during starvation it uses very little albumin compared with fats and carbohydrates. But in these cases of the formation of fat from proteid in the diet we do not refer to the already organized proteid, which has become an integral part of the cells, but we refer to the circulating albumin which has recently been taken in in the food.

The other food substances can be formed from proteid, but proteid can not be formed from carbohydrates or fats: it must be contained as such in the food. Only plants can form albumin or proteids synthetically from simpler substances.

Fats and carbohydrates may, it is true, take the place of albumin to a large extent; for example, if we place or express the amount of albumin that a dog requires to maintain the balance of metabolism (nitrogen equilibrium) on a diet of proteid exclusively,—at 100 per cent.,—it is possible to reduce the albumin breakdown to 6 per cent. by an abundant substitution of carbohydrates in the food, but even if we increase the ingestion of carbohydrates beyond the amount of potential energy required for the animal, the albumin breakdown can not possibly be reduced further than 6 per cent. of the amount required when an exclusive proteid diet is given. The fact that a superabundance of calories is given in the carbohydrate ingestion and still an albumin breakdown of the incorporated or organized proteid can not be prevented, seems to show that this is not due to any deficient supply for caloric requirements of the animal—an animal may be fed ever so abundantly with carbohydrates and fats, and it will eventually, though much later than if it had received no food whatever, die of albumin starvation. Gelatin, though it is closer related to albumin, proteids, or fats than carbohydrates, can not replace albumin in the food. Gelatin can not, therefore, give rise to proteid; it may, like carbohydrates and fats, substitute albumin to a certain limit, but beyond this limit—that which I have described as the 6 per cent. limit—there is nothing that can replace albumin. The effect of albumin beyond this limit is not a caloric, but a specific, one. By this I mean that this 6 per cent. of the total amount of albumin that is required to maintain the nitrogen balance is necessary, in some inexplainable way, for life, but not for purposes of producing heat, tissue construction, or force-production. So, by abundant fat and carbohydrate feeding, we can replace the albumin or proteid consumption and reduce the amount necessary to 6 per cent. of that which would be required if we fed on proteid or albumin and nothing else. We can get along without fat and carbohydrate in the diet for a time provided we can feed on proteid, but we can never entirely dispense with proteid.

When dogs are fed on nothing but the leanest meat, unless excessively large quantities are given they rapidly lose flesh. The same is true when we attempt to maintain the nitrogen balance or normal proteid percentage of the human body by an exclusive proteid diet: it can be done, but it requires enormous quantities. It might be supposed that it would be necessary to supply only the amount of nitrogenous food, proteid, or albumin which an organism uses up in a state of starvation in order to maintain its proteid balance (by proteid or nitrogen balance is meant the state of the body in which it neither gains nor loses albumin).

When an animal is fed on an *exclusive* proteid diet, giving daily just the amount which is burned up when a mixed diet is given, the animal will continue to use his own organized proteid and will lose weight. Suppose a dog loses fifty grains of albumin a day during starvation, and we feed him on albumin by supplying fifty grains of it—just the amount he burns up, nothing more. He will still keep on consuming his incorporated or already organized albumin. If we add a further amount of albumin,—say fifty grains more,—the proteid consumption will jump far beyond this amount, and this will occur every time we add new amounts of proteid—again and again on increasing the proteid food the amount burned up will exceed the amount ingested. For a long time the albumin breakdown will always exceed the ingestion. Finally, but late in the experiment, the albumin balance is secured.

Exclusive ingestion of albumin, then, is a very bad means of preventing loss of albumin from the system. Such large quantities of it are necessary that the digestive organs, or at least those of human beings, can not assimilate them for any length of time. Apparently this easily decomposed substance is consumed so rapidly by the oxidizing processes of the body that it has no opportunity for organization—that is, for a transformation to that form in which proteid is deposited in the tissues. If, however, we add carbohydrates or fats (also gelatin) to the proteid ingested in the body, the condition at once changes. The decomposition of the proteid at once decreases, so that the loss of proteid becomes less or even ceases, according to the amount of other substances added. The loss of proteid may even be converted into a gain or deposition of proteid. This is usually caused by the economizing effect of these substances upon proteid. As to the manner in which this economizing process is carried out, we may assume that the oxidizing forces of the organism are, in the latter case, so much occupied or engaged with these other substances that a portion of the albumin may escape them. This assumption is also in accord with the fact that carbohydrates and gelatins, as the more easily decomposable substances which have a certain greater affinity to the oxidations of the body, also exert a greater saving effect and draw the course of the decomposition further away from the proteids than the less easily decomposable fats. This difference between fats and carbohydrates in regard to their saving effect toward proteid is manifestly of great importance for nutritional therapeutics.

Proteid is absolutely necessary then, but by itself will not suffice. The question has been raised by Moritz (*l. c.*, p. 21) and others whether we can limit ourselves to giving, besides the proteid, fats exclusively or carbohydrates exclusively, or must both be present? If the latter is the case, in what proportion must it be? And also whether it is advisable to diminish the decomposition of proteid considerably by means of large quantities of fats and carbohydrates, so that but little proteid is necessary in the food, or is it better to make the ingestion of proteids abundant?

Only practical experience can give a satisfactory answer to those questions, and experience shows us, in the first place, that human beings will always consume all three of these food-stuffs if the climatic conditions permit it. Milk, also, the exclusive nourishment of the suckling, contains all three of these elements. Experience also teaches that the average consumption of proteid is fairly large everywhere except where the people are under compulsion or suffer from want, as when the crops fail. The remainder of the nourishment is divided between the fats and the carbohydrates, the latter getting the larger share. The well-to-do classes consume rather more fats and, correspondingly, less carbohydrates than the poorer classes. Nevertheless, the consumption of the starches is greater than that of the fats.

On the basis of extended experiments as to the constitution and composition of the food of a laborer weighing 70 kilograms (144 pounds) and performing moderate work, Voit has fixed upon 118 gm. proteid, 56 gm. fats, and 500 gm. carbohydrates as a normal requisite. For the present the absolute figures interest us less than their relation to one another, and especially the relation of the albumin to the nonnitrogenous substances, since the absolute numbers may vary with the bodily weight of the individual in question.

Concerning the relation of the proteids to the nonnitrogenous substances, there are great differences of opinion. The quantity of proteid stated by Voit has been called too large. This was based on experiments in which the amount of constitutionally assimilated proteid in a man had been preserved in cases where much smaller quantities of albumin were given than those stated, and then they were supplemented by greatly increased quantities of substances which economized proteid.

The diet proposed by Voit contains an amount of energy represented by the proteid approximating 16 per cent. of the total energy contained in the food. This can easily be ascertained by the caloric calculation. Moritz collected a large number of statements regarding the composition of the food of persons belonging to different classes and performing different work. From these he calculated the energy represented by the proteid. It corresponded fairly well with the amount calculated by Voit, being 17.3 per cent. Thus about one-sixth of the total energy of the body is given to it in the form of proteid. When we consider how appropriate everything is which man does instinctively, we can not consider this fact accidental and subordinate. The figures given by Rubner in his scholarly article in the "Handbuch d. Ernährungs-Therapie," Bd. 1, S. 20, entitled "Die Physiologie d. Nahrung u. d. Ernährung," and those of Atwater in the publications from the United States Department of Agriculture, support the views of Voit just given.

Man tries to vary his diet as much as possible. It is, therefore, interesting to observe that this mixed diet generally maintains a similar relation between proteid and nonnitrogenous substances, even when the diet is varied, when it is taken both from the animal and from the vegetable kingdom, and when it is occasionally varied in regard to single articles of food. Probably the explanation of the phenomenon in question may be found in this fact. According to Moritz (*l. c.*), we must refer the average proteid consumption of human beings to the natural conditions which govern nourishment in his environment. We know, however, that influences which were accidental originally may exert a very great influence over the organism if they continue long enough. The organism gradually adapts itself more and more to these conditions, until it is finally fully adequate to them. When this occurs, these conditions, which were formerly new, now in turn become necessary to it. Moritz suggests that this consideration should make us cautious about withdrawing a man from conditions of nourishment which have lasted for ages, and putting him on a diet which is rich in fats and carbohydrates and poor in proteids, simply because we know, on theoretical grounds, that a great portion of the proteid decomposed in the body serves for calorific purposes, just as well as fats and carbohydrates ("Die Grundzüge der Krankenernährung," by F. Moritz, p. 23).

An abundance of proteid in the organism is the necessary groundwork for a good condition of all organs that are rich in protoplasm, and more especially of the muscles. If, however, the proteid shall be deposited abundantly and a plenteous supply of proteid maintained, the amount of proteid in the food must not be small. Experiments in metabolism have shown that the more muscular a man is, the greater is the transformation of proteid and the demands for its presence in the food.

It is also possible that a definite mixture and relation between the albumin and the other oxidizable material of the food is especially adapted to the organs which are concerned with the combustion of the food substances. This has been compared to the practical fact that there are furnaces the construction of which is suitable for a particular kind of fuel. We must also consider that certain intermediate products of the proteid metabolism may be of use to the organism. In this connection attention might be called to the great number of substances which are found in the urine and which, for the most part, must be considered as the intermediate and by-products of the decomposition of the food and which probably play some definite though unknown rôle in metabolism.

It should also be remembered that the demands which the various food substances make upon the digestive organs are not the same; the proteids, as well as the fats and carbohydrates, require special functions. It is, therefore, also possible that here, at the entrance gate of the organism, a definite mixture of the food-stuffs will be the best in the long run if this mixture employs all the functions equally and does not overload and overtax any of them. A diet which is too rich in carbohydrates usually is very voluminous; one which is too rich in fats usually is hard to digest and excites repulsion. In short, there are many reasons which may be adduced in support of a relatively large consumption of proteid, such as is seen in the general mode of living.

In considering food materials, at least in so far as they are intended to furnish a perfect nutrition, we probably are also justified when we devote our attention not only to their amount of energy in general, but also more particularly to their proteid contents.

The question whether the animal or vegetable character of a food material has any specific influence on its nutritive value is of practical importance. To be sure, the three great groups of food-stuffs are represented in the same manner in both natural kingdoms. In regard to their chemical activity, however, their representatives have partially different properties, so that it is possible, on this account, that they may not all be adapted for nutrition in a like degree.

It has, however, been demonstrated that the vegetable substances act in the same manner upon the animal metabolism as the animal substances. Vegetable matter containing proteid may maintain and increase the amount of proteid in the human organism, just as animal substances and vegetable fats play the same rôle in our metabolism as animal fats. As to the carbohydrates, they, of course, are taken almost exclusively from the vegetable kingdom. There are a number of dietetically important considerations as to utilization, digestibility, cheapness, secondary results, etc., according to which the animal and vegetable food materials differ from one another.

The problem of nutrition in practice fortunately becomes easier than one might have expected from the great number of theoretically important points of view which are presented by special workers on this subject. From time immemorial the human race has instinctively mixed its diet so as to contain the three fundamental food principles, but the civilized races have mixed them in such a manner as to contain the proper proportion of these materials. It is interesting to note that as we vary the food materials to a sufficient degree we insensibly hit upon a considerable proportion of proteid and also upon an appropriate mixture in the food. If we proceed qualitatively in the ordinary paths which have the sanction of experience, and as to the rest apply only the calorific standard, it is usually sufficient to enable us to attain a suitable diet. We can not thus avoid smaller differences in the composition of the food, but that is of no importance. In any extended period of time they always equalize themselves, and the organism adapts itself very well, within certain limits, to a varying introduction of food materials, with its corresponding decompositions. In the preference which is shown, in the diet of private patients, for certain animal substances rich in proteid, such as milk, meat, and eggs, we are, as a rule, also insensibly brought to a diet which approaches in general the diet of the well-to-do classes.

The selection of a diet for large bodies of men, such as for hospitals, asylums, and armies, is a different problem. Here the normal values must be fixed and definite, once for all, and we are also dependent upon economic considerations. Here it is imperatively necessary to consider the nutritive value of the various food materials as shown by chemical analysis, and more especially to see to it by calculation that, so far as a complete nourishment is concerned, the diet not only satisfies the demands for energy of the body quantitatively, but also maintains the aforesaid proportions in the mixture of the various food substances.

Chemical analysis of food materials is not an absolutely accurate basis for calculating their nutritive value. We must consider also the degree of utilization of the various food materials if we wish to learn the real amounts of the food substances which are utilized for metabolism. It is well known that the entire nutritive matter of a food material is not really taken up by the body, and that a definite portion, which varies according to the constitution of the food material, is passed out with the feces. Indeed, there are differences between the single food materials.

The resistance which a food material offers to the resolvent efforts of the digestive juices is the chief determining factor in regard to the amount utilized. This is apparent, since the more abundantly a food substance can be dissolved from the food material in a liquid form in the digestive canal, the more it will also be absorbed. The physical constitution of the food is of especially great importance in this regard. The more compact a food material is, and consequently the more difficult it is for the digestive juices to penetrate it, the less will its solubility be and the slighter its absorption. On the other hand, the more porous and loose its structure is, and consequently the easier it is for the digestive agencies to penetrate, the more complete is its utilization.

The mechanical constitution of foods, the degree and kind of consistency—this, as a rule, is the deciding factor for the amount of work which any food material imposes upon the digestive organs. For instance, it has been proved that compact rye bread is utilized less completely than light wheat bread ; that boiled, unground lentils are utilized less completely than lentil flour, and pieces of potato less than mashed potatoes, etc. In the next place, the length of time which the food materials stay in the intestinal canal is a determining factor for the completeness of the absorption. If this time is comparatively short, such as is the case if the food exerts a stimulating effect upon the peristalsis or if diarrhea exists, the utilization is injured.

For the great majority of the more important food materials the extent of the utilization has been determined by a comparative chemical examination of their composition and by an examination of the feces after any particular food has been eaten. As stated elsewhere (see chapter on Examination of the Feces), this method is not entirely accurate, since, according to recent investigations, the feces in all cases consisted to a large extent of thickened intestinal secretion—often, indeed, almost entirely. In practice, however, we would not err if we assumed the feces to be the remainder of the food conditioned by an incomplete utilization.

Most of the conclusions drawn when only one food material was ingested differ from those, however, when various food materials were taken simultaneously, because these may mutually affect each other in their utilization either favorably or unfavorably. We can not, therefore, transfer the aforesaid values of foods to a mixed diet offhand if we wish to be accurate. Besides, the experiments were usually made with very large quantities of food material, whereas it is well known that smaller quantities are utilized more completely.

For a calculation of the amounts of energy arising from the food materials for the body it would plainly be too circumstantial to make a reduction in the nutritive contents of each individual food material until the really utilized amount is reached. Therefore other caloric numbers are given for the food substances. These figures are intended to do away with this calculation, and are smaller than the former ones, on account of the average loss in absorption. Thus, for 1 gram proteid we put 3.2 calories (instead of 4.1); for 1 gram fats, 8.4 calories (instead of 9.3); and for 1 gram carbohydrates, 3.8 calories (instead of 4.1). In practice these figures are very convenient, but, of course, have the value only of approximations.

Besides its importance for a judgment as to the real nutritive value of food materials, the knowledge of the utilization of foods is of interest for us also because it gives us information as to the amount and composition to be expected. Whereas those food materials which are incompletely utilized, especially the vegetables, usually give copious, watery, and often pulpy stools, those which are more completely utilized give smaller and firmer stools and are often followed by constipation. Therefore the physician must have some knowledge of these conditions.

The United States Department of Agriculture has given valuable encouragement to scientific investigations on the utilization and digestibility of various American food-products. The new respiration calorimeter devised by W. O. Atwater (Bulletin No. 63, United States Department of Agriculture, Washington, 1899), a most ingenious apparatus, has been used to determine the caloric values of various foods and their effect on metabolism.

The reader is referred to the publications of the United States Department of Agriculture for further information on these subjects, especially to the following:

" Losses in Boiling Vegetables, and the Composition and Digestibility of Potatoes and Eggs," by H. Snyder, Almah H. Frisby, and A. P. Bryant. Bulletin No. 43, 1897.

"A Digest of Metabolism Experiments, in which Balance of Income and Outgo was Determined," by W. O. Atwater and C. F. Langworthy. Bulletin No. 45, 1897, and the many investigations on nutrition and dietary studies published by this department.

For the effect of alcohol on digestion the reader is referred to my work on "Diseases of the Stomach," second edition, page 296.

THE DIETARY PROPHYLAXIS AND HYGIENE.

DIET DURING EPIDEMICS OF CHOLERA, TYPHOID FEVER, AND DYSENTERY.

Fundamental Rule.—Nothing raw or uncooked is permitted; nothing that has been exposed to the air; that has been much handled or that may have come in contact with unboiled water. Every digestive disturbance is to be carefully avoided.

General Rules.—The drinking-water of the locality in question is the greatest source of danger; therefore all water which is used (whether for drinking, for cleaning the food or the dishes, for rinsing the mouth, for washing, for bathing, etc.) must be boiled for fifteen minutes before use, and kept in the vessel in which it was boiled until used. Besides this, water intended for drinking or rinsing the mouth should be slightly acidulated, half an hour before using, by means of hydrochloric acid or citric acid; it is sufficient if the acid test can just be detected. The acidulation is best done in large quantities, and the test for it is blue litmus paper, which must change its color to red.

If, in some cases, it is impossible to boil all the water, then that intended for external use, as for washing, should have soft soap added to it half an hour before using. Finer soap may also be used, but its results are not certain. Rain water, collected under personal supervision, should be used for washing, if possible; the tubs, etc., must not previously have been cleansed with ordinary water, or, if they have been thus cleansed, they must first be filled with boiling water.

Artificial soda-water or Seltzer water is just as dangerous as ordinary water.

Ice is equally as dangerous, and should, therefore, never be used in drinks; indeed, it is best to avoid cold drinks altogether. If, however, ice is made of distilled water, or water of known purity which has come from a distance, and is therefore not open to suspicion, it may be allowed.

Everything which *may have* come in contact with water or with the fingers of a third person is dangerous; also everything which has been exposed to the open air, since the germs are disseminated by the air and by flies. Thus, one should avoid especially all milk, vegetables, fruit, fish, meat, cheese, butter, cold roast meat, confectionery, bread, rolls, and the like, which have been exposed to contamination. All these may be eaten only after they have been boiled, thoroughly roasted, sufficiently heated or acidulated, or disinfected in some definite way or other. Confectionery should never be bought in the store after it has remained on the counters for some time; likewise cake so exposed should never be bought. Bread and rolls should be bought as fresh as possible, even warm; they should be bought at the bakery, and heated over in a bake-oven for several minutes at roasting temperature, or exposed on all sides to the direct rays of the sun for half an hour. If one wishes to be absolutely safe, it is well to wipe off the crust with bread-crumbs, which are then burned, or even stop eating the crust altogether. It is also well to toast the slices of bread thoroughly. * All these foods should be eaten as warm as possible, and immediately after being prepared.

The food which is to be kept stored must be tightly covered and kept in a meat safe, in a quiet, well-ventilated place, such as the cellar or pantry. It should never be kept in the kitchen or in an inhabited room.

One should avoid all foods and drinks which are hard to digest, and which one has found, from experience, do not agree. One should also avoid overloading the stomach and all gluttony, carousals, and iced drinks.

---

* None of the above-mentioned foods are injurious in themselves. Persons owning a garden, with vegetables and fruit, or those that can get cheese with the tin-foil unopened, or caviar in the original package, or whose bread is baked in the house may consume all these foods without any bad effects if they are prepared properly and they agree with them. Personally I am opposed to the use of any form of cheese during the prevalence of intestinal diseases.

Before all meals, and also before smoking, the hands should be washed thoroughly with water, soap, and a brush, special care being taken to remove the dirt under the finger-nails. The mouth should be rinsed and brushed, using slightly acidulated water or a carbolated dentifrice.

As far as possible no foods or drinks should be taken in public places, restaurants, or hotels, nor in other people's houses. In such cases one must choose substances in the original package, as natural mineral waters or strong alcoholic drinks, as wine or brandy, or such as have been boiled, as coffee or tea. The foods to be chosen are such as have been raised to a high temperature and are still warm, or such as have been treated by acids, or soups, boiled meat, roasts, baked potatoes, boiled vegetables, eggs, may be practically considered sterile, but *not* cold roast, rolls, salad, etc. As few dishes as possible should be used, and in hotels one should drink in his own room and directly out of the bottle. The fewer the vessels or dishes used, the fewer the sources of infection.

SPECIAL DIETARY PROPHYLAXIS.

Soups.—No unboiled water is to be added to the soups which are done or nearly done.

Meat.—All boiled, steamed, stewed, roasted thoroughly, soured, or kept in the unbroken original package, also all preserved, salted, or smoked meat when in the entire piece, such as corned beef, ham, smoked meat. The outside or enveloping layer of large pieces of meat should be cut off and burned up. I advise avoiding sausages of all kinds.

No unboiled water must be added to the sauce when ready or nearly done. What is left over from the meals must not be kept in the open, but well covered and protected.

Large pieces of meat are not heated through and through as certainly as small ones—two to three pounds should be the limit.

Fish and Shell Fish.—Directions the same as for meat. During epidemics of typhoid, dysentery, diarrhea, etc., oysters are best avoided, not because they may be a source of infection, but because they readily decompose in the intestine when a state of putrefaction already exists there.

Eggs.—All styles, including raw. The mouth should not be placed on the egg for sucking it.

Fats.—Lard, bacon, oil, melted butter, lard butter. Fresh butter and oleomargarin only if imported from localities where there is no sickness ; it is best, however, not to use them at all if their origin is not definitely known.

Baked Foods.—Should be baked in the patient's own house. Otherwise bought as fresh as possible, rubbed off with hot sterilized towels, or exposed to the sun, baked over, or toasted. To be kept in a cupboard with glass shelves.

Puddings.—All styles. Only boiled water to be used in the sauces.

Vegetables.—All boiled, stewed, or preserved. Raw vegetables (salads) should be avoided.

Cucumbers contain practically no food material, and can therefore be dispensed with even if they were wholesome, which they are not, as a rule.

Desserts.—Made of wine, rice, boiled fruit, hot cup custard, or boiled milk. The sauces to be prepared with boiled water or milk only.

Fruits.—All boiled, stewed, roasted, or preserved in the patient's own house. Fresh, evaporated, or candied fruit can be eaten without further preparation only if they were picked before the epidemic, or in a healthy country and bought in the original package (bag, glass, box, or can), and thus did not come in contact with hands, scales, or bags. Such fruits as imported grapes, apricots, peaches, raisins, currants, dates, dried fruit, and preserves. They may also be eaten if they are consumed without their hulls—that is, nuts, chestnuts, oranges, melons, and carefully cleaned and pared apples and pears. All fresh fruits may be eaten raw if they are picked by people with clean fingers, in uninfected gardens controlled by healthy people, and are immediately packed in vessels which have not come in contact with unboiled water (baskets, plates, or dishes) and delivered.

During epidemics of typhoid and dysentery, and for troops operating in tropical countries, I advise the avoidance of fruit altogether. Perfectly healthy persons and convalescents may be permitted to eat stewed fruit (apples, pears, apricots) while the stew is still hot, and if the dried fruit has been brought from the native country in sealed packages.

It is senseless to destroy all fruit in times of epidemics. The important matter is first to prevent all contact between the fruit and water, suspected hands, and vessels; secondly, not to cause digestive disturbances by eating too much or unripe fruit.

Drinks.—Boiled water and milk, condensed milk in sealed packages from healthy regions, natural mineral waters from regions where there is no sickness (Saratoga Vichy, Rockbridge Alum, Poland, Harz, Sauerbrunnen, Apollinaris, Bellthaler, Niederselters), all tea, coffee, chocolate, cocoa, wines, cordials, brandies, beer from healthy regions. All these may be drunk only out of glasses and cups which have been washed with boiled water.

In a German work on diet I observe the recommendation of home-made wine. Personally I am loath to permit wine made at home—not because it may constitute a source of infection, but because very few persons understand the manufacture of wine at home. It is almost impossible to obtain the requisite vats and implements and to permit of proper aging of the wine. In my experience the much-praised home-made wines are more intoxicating than wines bought from established vineyards, because they still contain the higher toxic alcohols (methyl, propyl, butyl, and amyl alcohols), which are very deleterious, even in the smallest doses.

Prohibited.—All unboiled water, unboiled milk, buttermilk, cream, curds, fresh butter, fresh cheese (if one does not own a cow or does not get milk and milk-products directly from a healthy region), all raw vegetables (especially unacidulated salads, cresses, radishes, turnips, onions, garlic), and fruits the origin of which is unknown; all cakes and sweetmeats from stores; raw meat, raw and also roasted sausage, cold sliced meat from stores (sausage, ham, smoked meat, fish, and roast), caviar that is exposed to the air or that is imported from epidemic regions, mussels, and, for precaution, also oysters.

Furthermore, everything which one has found out from experience does not agree, especially all heavy, fatty, inflating foods,—rye bread, fresh bread, pumpernickel, pork, cabbage, turnips, cucumbers, poached eggs, eels, hard-boiled eggs, puddings,—and also unripe fruit, overloading of the stomach, cold drinks, carousals, and gluttony.

Advice.—Live carefully and moderately. Masticate well and eat slowly. Eat and drink only at home. Go to bed early and rise late—sleep as long as you can.

If diarrhea occurs, it is to be taken much more seriously than usual. The physician must be called at once, and the water-closet must be disinfected according to his directions.

INTESTINAL INDIGESTION (DYSTRYPSIA).

General Rules.—A regular mode of living and much rest. Eat every day at the same time, either often and small quantities at a time, or less frequently and in larger amounts, just as best suits the patient. Eat slowly and masticate thoroughly. The food must be neither cold nor hot, but lukewarm. Rest before and after meals, and do not oppress the stomach after meals by tight-fitting clothes. The mouth should be cleansed and disinfected after eating.

Bill of Fare.

Soups.—Few, thin, and containing little fat. Lean meat soup, made also of meat-extracts, with the yolks of eggs, grits, meat-balls, and few herbs, which must be strained out later; few noodles, rice, barley, sago, and the like; beef-tea, strong broth, Liebig's cold broth, fruit soup, wine soup, beer soup, strained potato, pea, bean, lentil, and bread soup.

Less frequently, thin, likewise strained, water gruel and milk porridge of grits, rice, flour, etc., curds, and buttermilk soup.

Meat.—Soft lean meat of young beef, of calf, mutton, deer, partridge, pigeon, chicken, turkey (the white meat), also game, tongue, calf's-brains, sweetbreads, when properly prepared. They are best boiled, set on with hot water, or sliced thin, also slightly roasted, thoroughly roasted, or boiled; also finely cut up fresh ham, juicy fresh smoked meat, cutlets without the bread-crumb dressing, meat jelly, ragout. No fatty sauces.

In case of necessity: Armour's soluble beef, which contains from 30 to 35 per cent. of actual nutritive substance, malted milk, somatose, nutrose, fifty grams twice daily, in soups, rice, or between rolls;

meat-juice, as Valentine's, Hamburg's, Brunn's (Wiesbaden), Wyeth's, etc.

Fish and Shell Fish.—Trout, pike, sole, codfish, haddock, plaice in small quantities. Anchovies, sardines, herring, whether pickled, smoked, or prepared, sliced salmon, caviar. I advise avoiding crabs, lobster, and crawfish.

Eggs.—Raw, soft-boiled (up to three minutes); also finely chopped hard eggs with abundant salt; the yolk beaten up with sugar; also with strong wine, rum, cognac; in soup, with beer, milk, water.

Fats.—Caution. Good butter on wheat bread, toast, and together with a little beef-suet; also in the foods; no sausages; no fatty sauces.

In the case of fats it is especially important to determine by experiment which kinds agree with the patient.

Baked Food.—Of wheat flour: toast, cakes, biscuits, rolls, wheat bread, especially stale and toasted (toast). Often, however, the opposite is true—that Graham bread, whole-meal bread, and rye bread agree better with the patient.

Puddings.—Small amounts of rice-milk, chocolate, orange, apple, sago, groats, and millet pudding, and the like—light foods without any fat; Hartenstein's leguminose, maltoleguminose, and also pudding might be tried experimentally.

Vegetables.—Not much. Best avoided. The least harmful are spinach, salad, cresses, cauliflower (without the white sauce), tomatoes, asparagus, celery, mashed potatoes, fresh green peas (masticate well and afterward drink some water).*

Desserts.—Preserves, milk, fruit, or wine jelly, chocolate, curds (fresh white cheese) with milk and salt.

Fruits.—Best avoided. Ripe fruit (no sweet almonds or chestnuts). Evaporated and boiled fruits are safest (prunes, apples, pears); roasted apples; also candied and preserved fruits.

---

* **Edible Fungi.**—C. H. Peck (Report of New York State Botanist, 1895, p. 113) has asserted that fungi constitute a "very nutritious and sustaining diet," an opinion not confirmed by the careful experiments of Lafayette B. Mendel ("Jour. Phys.," vol. 1, p. 225). Edible fungi contain from 75 to 90 per cent. of water, and an average of total solids of only 10.5 per cent., and could contain, as a possible maximum, only 3 per cent. proteid, which, according to my analyses of stools made with the Boas stool-sieve after weighed amounts of *Morchella esculenta* had been ingested, is not even utilized, and therefore appears difficult of extraction by the digestive process. I conclude, therefore, that fungi are simply a dietetic "ballast" and had best be excluded from the diet altogether.

I regret to state that foods are frequently forbidden by general practitioners on purely empirical grounds. For instance, in one American publication I find all fruits and vegetables forbidden "because (1) they are generally plucked unripe; (2) because they are often in a state of partial putrefaction; (3) because they are often eaten in excessive quantities." Here the thinking dietarian must naturally ask, (1) What objection can there be to eating only ripe fruit; (2) fruit that has not undergone putrefaction; and (3) only the correct amount? In all cases it is important to watch the stools and study them by means of the Boas stool-sieve. If foods are digested and it can be proved that this is the case by the stool analysis, there is no reason for forbidding them.

Drinks.—Water (especially one hour before meals), milk, almond milk, buttermilk, kumiss, if there is no diarrhea, kephyr (an intoxicating drink made by putting kephyr fungi in milk), thin hop beer, light white and red wines, corn and acorn coffee and cocoa, oilless cocoa (boiled in water), and tea. Good claret or Burgundy (six years old), French or German importation, mixed with sterile water. Cider is best avoided.

Prohibited.—Fat meat or fish, sausage, fat sauces, very much fat in any form, bread-crumb dressings, whipped cream, fritters, eggs and bacon, poached eggs, tough, boiled-out meat, smoked meat, chipped beef, old ham, sturgeon, solid flour foods prepared with much fat, cakes, pancakes, potato cakes, pumpernickel, fresh bread, pastry, onions, carrots, turnips, cabbage, whole potatoes (roasted potatoes, after being scooped out of the peeling and mashed soft on the plate, are digestible and should not be forbidden), peas, beans, lentils, nuts, almonds, chestnuts, ice-cream and water-ices, cold drinks, brandies, heavy wines, strong coffee or tea, pepper, mustard, curry, and other strong condiments.

Advice.—Cold rubbing, especially of the hypogastrium. During hot weather a stay at the seaside or in the mountains of Vermont, New Hampshire, and Pennsylvania, near Pen Mar, Md., Buena Vista, Bedford, Pa., Hot Springs, Va.

Treatment with Natural and Artificial Mineral Waters.—See Hemmeter, "Diseases of the Stomach," second edition, pages promenading, if possible. Anticipating the calls of nature every day at the same time, even if no desire exists (best after a breakfast with a glass of Bedford magnesia, Saratoga, Congress, or Hathorn water, black bread and butter). The patient should massage his abdomen, starting at the bottom on the right side, then straight up and across to the left, and then down. This should be repeated for fifteen minutes. The leather-covered cannon ball weighing five pounds is excellent for this massage. Also seashore surf-baths.

Patients suffering from hemorrhoids should receive special treatment therefor and use very soft toilet paper (see chapter on Diseases of the Rectum).

Treatment with Natural or Artificial Mineral Waters.—*To be taken only when the diet does not suffice, and only under the direction of a physician.* Bedford magnesia, Saratoga, Hathorn, or Congress, sulphatic water (Bitterwasser) of Seidschitz, Friedrichshall, Ofen (Hunyadi János), Mergentheim (Karlsquelle), every evening one glass (150 to 200 c.c.) before retiring, and in the morning, on an empty stomach, one or two glasses; thereafter a walk of a half-hour is to be taken, the amount gradually to be increased; Carlsbad Schloss and Mühlbrunnen, Marienbad, Ferdinands, Franzensbader Salzquelle, every morning one or two glasses on an empty stomach, gradually increasing to six glasses. Only in case of need one or two glasses in the evening, two hours before or after supper; or one-half to one tablespoonful of artificial Carlsbad salts in a large cup of warm water, to be taken in the morning before eating.

Supplementary treatment, or, in the case of debilitated persons, at first the American chalybeate waters (see special chapter), then Schwalbach, Marienbad, Carolinen, Ambrosiusbrunnen, Franzensbad, Franzensquelle, Kissingen, Ragoczi, Pandur, Homburg, Elizabeth, Kaiserbrunnen, Cannstadt Sprudel, and Inselquelle, every morning from one to three glasses before eating.

Personally I feel it my duty to add that while the internal use of certain mineral waters is an undoubted adjuvant in the treatment, I have not seen a single case of cure of chronic constipation by their use alone. For methodical treatment, from which permanent success can be hoped, see chapter on Constipation.

SEVERE ACUTE INTESTINAL CATARRH (DIARRHEA), DIARRHEA WITH VOMITING, CHOLERA, ACUTE DYSENTERY, ACUTE INFLAMMATION OF THE APPENDIX.

It is advisable to eat only simple, strained gruel of grits, rice, flour, millet, barley, groats, sago, peeled barley, avenazia, mondamin, maizene, salep, old rolls, toast, prepared with some salt (not sugar), well cooked, and mixed with the beaten yolks of eggs; red-wine soup, containing well-boiled sago—called claret-sago soup; in this toast may be crumbled; well masticated. For drinks, warm, unsweetened indigenous teas, such as camomile, peppermint, and lime-tree blossom.

Sometimes, also, but only on the physician's prescription, beef-tea, strong broth, Liebig's cold meat broth, mulled wine, grog, red wine, whortleberry wine, southern wine, egg drink, coffee with egg, oilless cocoa, cooked with water and unsweetened, and sparkling wine. Also pieces of ice made of pure or, if possible, of distilled water; cold natural Saratoga Vichy, Seltzer water, Apollinaris, Harzer Sauerbrunnen, champagne.

Finally, according to circumstances, especially when the patient begins to improve, but only on the physician's prescription, warm boiled milk and milk porridge similar to water gruel, a slight amount of butter added to the soups, toasted slices of stale wheat bread and rolls, boiled rice, beef-tea, beer soups (boiled), boiled and strained whortleberries, the yolks of eggs beaten up with sugar, skimmed bouillon, a little sliced raw meat, finely chopped, slightly roasted, fresh, tender, young beef, and finely chopped or scraped tender lean ham (see "Dietetic Kitchen" in Hemmeter's "Diseases of Stomach").

In cases of need try, experimentally only, Nutrose, Soson, Armour's soluble beef, Valentine's or Wyeth's meat-juice. Somatose has a laxative effect.

Advice.—Woolen underwear, flannel belt; keep feet warm.

MILD INTESTINAL CATARRH (DIARRHEA), CHRONIC DYSENTERY, INTESTINAL TUBERCULOSIS, CONVALESCENCE, INFLAMMATION OF THE CECUM.

General Rules.—The food must preferably be liquid, mucilaginous, and poor in fats. Eat slowly and masticate and insalivate the foods thoroughly. They must be neither cold nor hot, but lukewarm. Do not eat much at any one time, but eat smaller quantities at shorter intervals (every three hours). In complicating gastric diseases base the diet on the results of test-meals.

Bill of Fare.

Soups.—Simple strained water gruel and milk porridge, prepared with salt, rarely with sugar, thoroughly cooked, of grits, rice, flour, millet, sago, barley, peeled barley, buckwheat grits, avenazia, farina, mondamin, maizene, stale toast, and rolls; also with the yolks of eggs and a little butter; a little skimmed meat broth or extract broth with the same additions; also with noodles and fresh herbs; red-wine soup (without the whipped white of eggs), wine sago soup; in case of need, also beef-tea, strong broth, Liebig's cold broth.

Meat.—But little, finely chopped, raw or slightly roasted, lean, tender, young, fresh (superlative) beef, thinly sliced, tender lean ham, broiled white meat of chicken and pigeon, and boiled and finely cut sweetbreads without fatty or butter sauce.

As the condition improves, boiled and roasted tender beef and mutton, white turkey meat, tender game (without haut-gout or the gamey flavor), such as deer (also mutton à la deer), stag, partridge; meat jelly, boiled calf's-brains.

Fish and Shell Fish.—Trout, blue fish, fresh salmon, perch.

Eggs.—The yolk raw, beaten up in soups, coffee, wine, sugar, etc.; the egg-white in water; soft-boiled eggs (boiled up to three minutes).

Fat.—A little good butter on bread or in soups.

Baked Foods.—Toast, wheat flour biscuits, cakes, old toast and wheat bread, best toasted, steeped, or well masticated.

Flour Foods.—Thin porridge of rice (with milk), sago, groats, mondamin, without fat.

Vegetables.—Spinach (without eggs), mashed potatoes, chestnut porridge.

Dessert.—Wine jelly, milk jelly (without fruit and cream sauces or whipped white of egg), curd (fresh, white cheese—Neufchâtel), with salt, rice pudding.

Fruits.—Later on, preserves or jelly of finely strained whortleberries, pears, preserved cranberries, cowberries; sucking out the individual fresh grapes and ripe oranges.

Drinks.—Not much at any one time. Indigenous teas (peppermint, camomile), acorn coffee, acorn cocoa, rice-water, thin barley, oats, thin watery infusion of cocoa (oil extracted); less suitable are very thin tea and coffee, with very little, if any, sugar; boiled milk, either pure or mixed with some water or with a small amount of cognac or rum; some red wine, claret, or imported Burgundy. Not much water, this preferably boiled, and with three ounces of red wine to every eight ounces of water or the above-mentioned substances added.

On a Special Prescription.—Cooled drinks, Seltzer water, Apollinaris, Saratoga Vichy (not when there is diarrhea), Burgundy (Beaune), brandy, chablis.

Prohibited.—All fats (except some butter on bread or in soup, where there may be a little kidney fat), also fat bouillon, sauces, meat, baked foods, bacon, sausage, whipped cream, lard, etc., all fish (except specified), lobster, crawfish, crabs, all firm, hard, boiled, smoked, salted, or preserved meat, poached eggs, eggs and bacon, all flour foods made with eggs and fat (pancakes, potato cakes, puff paste, flaky paste, cakes baked in lard), dumplings, fritters, rye bread, hot bread, confectionery, puddings, pastry, chocolate, fruits, vegetables (most of them), preserves, cheese, Moselle wine, fruit wine, beer, weiss beer, lemonade, cordials, brandy, cider.

Wear woolen underwear and flannel belt.

EXTRABUCCAL FEEDING. RECTAL AND SUBCUTANEOUS ALIMENTATION.

The esophageal, gastric, and duodenal abnormalities which constitute indications for extrabuccal feeding have been stated in the author's work on "Diseases of the Stomach," second edition, page 217; and in order to bring the entire subject under one heading, I have repeated these indications in the text of the following. The intestinal indications for extrabuccal alimentation are: (1) Any condition leading to severe intestinal hemorrhage; (2) intestinal obstructions; (3) intestinal stenoses; (4) some forms of prolonged appendicitis; (5) contusions, rupture, and perforation of the intestines; (6) conditions of great exhaustion in which the intestine can not digest the requisite amount of food, owing to an existing enteritis, dysentery, or other pathological conditions.

Extrabuccal alimentation may be carried on by three methods: (1) Rectal feeding by means of nutritive enemata; (2) subcutaneous or intravascular injections of food materials; (3) feeding after gastrostomy. There are cases in which the intestine reacts by pathological symptoms if it is obliged to take in a sufficient amount of food to maintain the normal nitrogen balance, whereas it does not react morbidly if a smaller amount of food than is requisite is introduced. In such cases a state of hyponutrition supervenes in which the patient continually loses weight, because there is a deficit in the amount of food material introduced. As the incapacity of the bowel to cope with the required amount of food is frequently only temporary (gastro-enteritis, dysentery, some forms of enteroptosis and intestinal ulcers), the deficit in the food can be made up by subcutaneous or rectal feeding, until such a time when the bowel shall have completely recovered from its temporary infirmity. Extrabuccal feeding here is valuable in tiding the patient over the critical stages of the disease; it is supplementary feeding because it may serve to bring the amount of food introduced up to the standard required for the nitrogen balance. It is not possible to maintain the nitrogen balance by extrabuccal feeding alone. Ewald ("Extrabuccal Feeding," "New York Medical Record," August 18, 1900) is of the opinion that in very much depreciated individuals, with a greatly decreased metabolic activity, the nutritive balance may be maintained by extrabuccal feeding exclusively. In these cases the metabolism is so depressed that it does not require very much to maintain it, and the entire metabolic activity of such patients will have to be increased before they can recover. Of the various types of extrabuccal feeding which I have enumerated, the only ones which are practical and of importance to the physician are hypodermic or subcutaneous feeding and rectal feeding. Feeding after gastrostomy has more of a surgical than a medical interest, and is usually carried out under the supervision of a surgeon.

Rectal Alimentation.—Of the various forms of enemata which are described in the following, those of Boas and Ewald are the most useful. The pancreatic meat enema of Leube and Rosenthal is inconvenient in practice, because it is difficult to obtain fresh pancreas and to prepare it in the manner suggested by Leube. Peptone and albumose solutions have been extensively employed as additions to enemata composed largely of eggs. It is risky, however, to employ peptone in form of rectal enema, because it frequently produces diarrhea if used in quantities exceeding fifty grams dissolved in 200 c.c. of water. It is rational to add a small amount of peptone to rectal enemata if it is desired to increase their nutritive value, but the solution should never exceed a strength of 25 per cent. of peptone. I have had abundant experience with nutritive enemata to which peptone and albumose have been added in amounts not exceeding the quantities stated, and have never observed a reaction of peptone or albumoses in the urine thereafter. It is not absolutely necessary that these predigested products should be added. I have given the evidence (John C. Hemmeter, "Ueber das Vorkommen von proteolytischen und amylolytischen Fermenten im Inhalt des menschlichen Kolons," "Pflüger's Arch. f. d. ges. Physiol.," Bd. LXXXI) that the sterilized aqueous extract of the rectal contents is capable of dissolving dried serum albumin and fibrin and of converting starch into maltose; from 62 to 75 per cent. of one gram of starch was converted into maltose by this extract under aseptic precautions. It is therefore probable, according to my experiments, that the pancreatic ferments survive in passing through the intestines. Therefore proteid and carbohydrates injected in form of enemata can undergo proteolysis and amylolysis in the rectum. Starches are better tolerated by the rectum than sugars. When more than from fifteen to twenty grams of sugar in 300 c.c. of liquid are injected, the rectal mucosa becomes congested and the enema is expelled by the irritation it sets up. A 50 per cent. solution of sugar may even set up a colitis, with excessive discharges of mucous diarrhea.

Concerning the **subcutaneous feeding,** it should be stated that it is now almost exclusively limited to the injections of sterilized olive oil. When dextrin is injected in this manner, it is usually excreted again in the urine. According to Leube (" Deutsch. Klinik," Bd. 1, S. 71), glycogen, when injected subcutaneously into animals, is not only promptly absorbed, but is utilized in the system and not excreted in the urine. The injection of carbohydrates is also very painful. Oil is much better suited for hypodermic medication and can be much more easily sterilized than glycogen, so that even if the latter could be readily obtained, it could not replace sterile olive oil for this purpose. I have had considerable experience with hypodermic feeding with sterile olive oil, on the basis of which I can confidently recommend it as a supplementary alimentation, not only in diseases of the stomach and intestines, but in all wasting diseases. It would appear that this mode of supplying the body with oil is far preferable to disturbing the stomach with cod-liver oil in pulmonary tuberculosis. In one of Leube's cases, as much as 200 gm. of oil, which equals 1860 calories, were injected under the skin and absorbed. I have frequently injected 100 gm. of oil in twenty-four hours, which would make 930 calories. If, in addition to this, we can give two enemata of 300 gm. of milk, 40 gm. starch, and 3 eggs daily, we would add 1100 calories, making a total of 2030 calories. Although the amount of food introduced in this way does not contain the daily normal quantity of albumin, nevertheless it contains about 60 gm. of egg-albumin. The total amount of nitrogenous substances contained in the carbohydrates and fats in this combined rectal and subcutaneous feeding amounts to 150 gm., corresponding to 1400 calories. Leube (*l. c.*) is of the opinion that complete nutrition of the body by these artificial means will eventually be possible. At present, however, with a very extensive experience with these methods of feeding, it must be stated that they can not replace completely the normal processes of feeding by the mouth.

In some cases of **feeding through gastric fistulæ,** undertaken for the relief of benign stricture of the esophagus, a sufficient amount of food was taken in to cause a gain of weight.

As a temporary supplementary expedient in transient infirmities of the intestine, extrabuccal feeding is very useful in tiding the patient over a transient digestive incapacity. Nutritive enemata are preferable to subcutaneous injections of oil ; they do not require aseptic precautions when administered and are absolutely painless.

The physiology of rectal digestion and the preparation of various enemata will now be taken up separately.

PHYSIOLOGY OF RECTAL DIGESTION.

In diseases in which the approach to the stomach is stenosed (carcinoma or stenosis of the esophagus or cardia), or in which the organ requires a temporary but absolute exemption from work (ulcer, cancer, and some forms of gastritis), or in cases in which the mildest and most digestible diet is not tolerated, it becomes necessary to support the strength by nutritive rectal enemata.

The history of the evolution of the nutritive enema is, from a physiological standpoint, very interesting; and as the author has at various times done considerable experimental work in this line, a brief review of the same is believed essential to a proper understanding of the subject.

The first to discover that the human colon and rectum absorbed an emulsion of eggs and water only when sodium chlorid was added were Voit and Bauer (" Zeitschr. f. Biol.," 1869, Bd. v). They found that these foods were not absorbed in the absence of salt, and in 1871 Eichhorst (" Pflüger's Archiv," Jahrgang IV, 71) confirmed their results. Injections of bouillon, milk, and eggs had been used long before this time, but no one ever attempted to ascertain to what degree the mucosa of the large intestine would absorb it. In 1872 Leube proposed a meat-pancreas injection ("Deutsch. Arch. f. klin. Med.," Bd. x, Reihe III), the plan emanating from the idea to transpose something of the character of pancreatic digestion into the large intestine. The preparation of this useful enema is as follows: Take 150 to 300 gm. of very finely scraped beef and 50 to 100 gm. of finely chopped pancreas of the calf or pig, and mix, with the addition of 150 c.c. lukewarm water, in a bowl; if desired, 25 to 50 gm. of fat may be added in the form of oil or butter; the injection must be made at the body-temperature.

This mixture digests thoroughly in the large intestine; its preparation is complicated, however, requiring very intimate mixing, and it rapidly decomposes. Leube reported a case which he kept alive six months by this enema exclusively, and in a similar way Riegel nourished a case of esophageal stricture for ten months. Ewald demonstrated that egg emulsion is absorbed without being peptonized or salted ("Zeitschr. f. klin. Med.," Bd. II), and Huber, while confirming Ewald's observation, added that the addition of salt or previous peptonizing really doubled the amount of emulsified eggs that was absorbed in the colon. Eggs, it must not be overlooked, contain a considerable amount of normal salt, and this may explain Huber's results (" Zeitschr. f. klin. Med.," Bd. XLVII) as to their absorption in part, even without the addition of salt. But it is an established fact that the addition of sodium chlorid very much increases the amount of eggs that is absorbed.

In a very interesting series of experiments Grützner offered a physiological explanation of this phenomenon (" Deutsch. med. Wochenschr.," 1894, No. 48), having demonstrated that under certain conditions particles of charcoal, finely cut horsehair, or sawdust impregnated with normal (0.6 per cent.) salt solution and injected into the rectum of rabbits, guinea-pigs, and rats, are found six hours later all along the small intestine, even in the stomach, while the rectum is empty. During a period of twenty-four hours preceding these injections the animals were starved. When the suspensions of these particles were made in distilled water, HCl solution, or potassium chlorid solution, instead of physiological salt solution, the particles did not ascend in the digestive tract. Grützner injected starch suspensions in normal NaCl solution into the rectum of human beings, and after a number of hours demonstrated starch grains in the gastric contents microscopically. Nothnagel first showed that sodium chlorid placed on the serous surface of the intestine is capable of starting antiperistaltic movements (" Beitrag z. Physiol. u. Pathol. d. Darms," 1884), and Grützner interpreted this observation as an explanation of the digestion of egg enemata containing salt. He assumed that the injected mass is moved upward through the entire small intestine, and so becomes digested and absorbed. Even Riegel (*l. c.*, p. 245) was satisfied with this interpretation, and added that it explains the negative results of Voit and Bauer without salt, and the positive ones with salt, and also those of Huber.

In my opinion the evidences that food substances move antiperistaltically upward in the intestine are not satisfactorily given in

Grützner's work. It is undeniable that minute particles of starch, charcoal, etc., are moved from the rectum toward the stomach in man; and I have been able to confirm this part of his results, as well as the fact that salt favors the ascent and HCl and KCl impede it. But this antiperistaltic motion I conceive to be only a very feeble marginal ascending movement, effected by surface contact of the particles with the epithelium, which in turn is moved by the muscularis mucosæ. This very slight marginal antiperistalsis is never visible to the eye, and can be demonstrated only by the progress of particles; it is not capable of propelling food masses; on the contrary, I have convinced myself that at the same time that the marginal peristalsis drags visible particles of charcoal toward the stomach with infinitesimal slowness, there may be an uninterrupted central current of food masses toward the anus. The marginal antiperistalsis may be a physiological thing, present at all times, and its object may be the raising or drawing up of portions of mucosa from one place to another in order to bring new surfaces in contact with the ingesta, or to replace a portion of surface to its normal topography after it has been dragged away from it by the downward current. The cohesion of these small particles with the mucosa can be seen when a piece of fresh animal intestine is sprinkled with lycopodium or finely cut horsehair particles and held under a gentle stream of water; the gut may be moved upward on the surface of the hand while the water moves downward, and still many of the particles will adhere. This illustrates the intimate mechanical adhesion of fine particles to the mucosa; their movement toward the pylorus is as yet not explained.

The antiperistalsis that Nothnagel produced by placing crystals of salt upon the serosa is quite a different thing, for it is plainly visible to the eye, and never occurs under physiological conditions (Nothnagel, "Erkrank. d. Darms," article on "Die Darmbewegung," S. 6, 1896). Among the abnormal conditions that may cause this visible antiperistalsis Nothnagel states stronger solutions of sodium chlorid and the introduction of food at an unphysiological entry, as which, in man, we must consider the rectum. He adds (*l. c.*) that from a physiological entry—*i. e.*, from the stomach—the strongest chemical irritants produce only peristaltic movements toward the anus. There are, then, two kinds of antiperistaltic movements—(1) those of Grützner being marginal, invisible, possibly physiological, and not capable of moving food masses, and (2) those of Nothnagel being visible, strong, and occurring only under abnormal conditions. Christomanos, a pupil of Nothnagel, has urged an objection against Grützner's results that seems to invalidate the conclusions of the latter—namely, he found (Christomanos, "Z. Frage d. Antiperistaltik," "Wien. klin. Rundschau," 1895, No. 12 and 13) that when his animals were prevented from licking up the expelled rectal contents, his results as to finding the particles in the stomach were negative. Dauber came to the same conclusion as Christomanos—namely, that the occurrence of particles of rectal injections in the stomachs of animals did not take place when they were prevented from eating their excrement. These objections are, however, set aside by Grützner's and my observations on the human subject. The experiments of Swiezynski ("Deutsch. med. Wochenschr.," 1895, No. 32) also confirmed Grützner's statements, in that lycopodium injected into the rectum was found in the stomach.

We must, however, again emphasize that this antiperistalsis is not capable of moving ingesta, and that it can not logically be taken as an explanation of the digestion of enemata.

The fact that normal salt solution favors the invisible marginal ascent of particles and that other chemicals impede it is perfectly natural. For if this antiperistalsis be physiological and be assumed to be going on at all times, normal salt solutions can not disturb it, for they are a physiological environment in which intestinal movements occur exactly as when the tissues are bathed in their normal juices. But HCl and KCl are chemical irritants to which the muscularis mucosæ reacts by efforts at expulsion.

The Occurrence of Proteolytic Ferments in the Colon and Rectal Contents.—The author's explanation of the digestion of egg and milk emulsions in the rectum and colon is quite different from Grützner's and from that accepted by Riegel and others, and is based on a very carefully conducted series of expriements on animals and human beings. We desire to speak only of actual digestion, for there is good reason for believing that albumin, fats, etc., may be absorbed from the large intestine as such, without digestion.

Without going into the details and technic of these experiments, we will briefly state the conclusions. After the rectal contents of dogs or cats are sterilized with saturated solutions of thymol and passed through a Pasteur filter, control cultures are made to ascertain that the watery extract of the excrement is sterile. For this purpose the meat peptone-gelatin and agar plates recommended by Nothnagel (*l. c.*, p. 22) are most convenient. The reaction of human excrement is generally weakly alkaline or neutral; very rarely, weakly acid under normal conditions (see chapter on Examination of Feces). This watery extract of normal rectal contents contains a substance which, in a digestorium (thermostat at 40° C.) and *in an alkaline medium* (equal to from 0.8 to 1 per cent. Na_2CO_3), *dissolves from 36.5 to 50 per cent. of Merck's dried serum albumin* in three hours under aseptic conditions. It will also digest fibrin, and has, in addition, a faint amylolytic power, converting from 10 to 14.5 per cent. of starch into maltose in an alkaline medium of 0.3 per cent. Na_2CO_3. I have not been able to find any fat-splitting action in this extract ("Pflüger's Arch. f. d. ges. Physiol.," Bd. LXXXI, S. 151).

As there are many bacteria that produce peptone in the breaking down of proteid, and others that ferment carbohydrates, the previous sterilization is necessary in order to exclude their action. Bacteria do not make peptone for any philanthropic purposes; the peptone they give rise to is an intermediate stage in a long series of decomposition products. It does not remain peptone, but is rapidly decomposed into amido-acids, ammonia, tyrosin, etc., and does not occur in the intestine as peptone pure and simple, but mixed with a number of other derivatives of albumin, some of which are proved to be toxic. This I state because even among medical men the opinion has been encountered that the bacterial peptone might be of utility to the organism in which it is formed. The feces for our purposes can not be sterilized by heat, because that would destroy any possible enzymes present.

It is certain, therefore, that rectal contents contain a proteolytic ferment; also one having a slight amylolytic power, acting only in a faintly alkaline medium, the action being destroyed in an acid medium. Whether these two digestive actions are carried out by one and the same ferment or by two different ferments we are unable to say. That it can not be pepsin is proved by the fact that it does not act in an acid, but only in an alkaline, medium.

It would be interesting to learn whether the walls of the large intestine secrete any proteolytic ferment. The colon of a dog that is kept clear of fecal masses by making an abdominal fistula and sewing it to the abdominal wall at the ileocecal valve secretes an alkaline fluid which has no proteolytic powers whatever, but there is an evident amylolytic ferment contained in it. The human colon can be plugged up in the transverse portion by introducing a balloon and inflating it; thereafter the part between the rubber balloon and the anus can be washed out with sterile normal salt solution. A secretion is formed in two to three hours, and can be collected on absorbent cotton placed in the rectum, and later squeezed out into a small beaker. The secretion is alkaline, but has, after filtration through a Pasteur filter, no proteolytic power. I have made the same observation on a human being having a fistula at the ileocecal valve. As the colon, therefore, secretes no proteolytic ferment, it is reasonable to assume that the ferment we have demonstrated is derived from the pancreas. In two patients with total atrophy of the gastric mucosa (atrophic gastritis), as evidenced by fragments of the mucosa found in the wash-water, the same proteolytic ferment was demonstrable in the colon contents. It was hitherto assumed that the ferments of the pancreas were destroyed in the intestine (see Rosenheim, "Die Erkrank. d. Darms," S. 46). A large number of similar experiments as above described justifies the belief, however, that trypsin, and perhaps amylopsin, may survive the passage through the bowel. Busch has shown that digestion may go on in the human intestine without gastric or pancreatic juice, without bile and secretion of Brunner's glands (Brücke's "Vorlesungen über Physiologie," Wien, 1885, S. 352). The patient on whom Busch experimented had received an abdominal injury by an accident, in such a manner that the gastric juice, together with the chyme, pancreatic juice, duodenal secretions, and bile, ran outward through a fistula. Thereafter Busch fed him through the fistulous opening communicating with the lower bowel, and succeeded in maintaining the nitrogenous equilibrium. He lowered coagulated albumin inclosed in small cotton bags into the bowel, and drew them out by a string five hours later, finding that from 5 to 35 per cent. of the albumin was dissolved. In Busch's experiments the action of the bacteria can not be excluded. The action of the succus entericus may explain the carbohydrate digestion, but as no proteolytic ferment could enter the small intestine, the digestion of albumin was probably due to bacteria.

In conclusion we may say that rectal enemata are digested probably by pancreatic ferments passing through the bowel, by bacteria, and by the succus entericus, which, even in the colon, has an amylolytic action; that certain foods—egg-albumen, fats, milk—can be absorbed as such without being digested. Grützner's marginal ascending motion of particles can not move ingesta antiperistaltically. F. Mall ("Johns Hopkins Hospital Reports," vol. 1, p. 70) holds that the propelling force of the intestines normally acts in one direction only; the antiperistalsis is found only as a pathological phenomenon, and all his efforts to force the intestine to work in the wrong direction by reversal (*l. c.*, p. 93) were negative.

PREPARATION OF RECTAL ENEMATA.

Indications and Methods of Administration.

Nutritive Enemata.—*1. Meat Pancreas Clyster* (*According to Leube*).—One hundred and fifty grams of good beef are scraped and then chopped fine; 50 gm. of fresh pancreatic gland, free from fat (either of a cow or of a hog), are mixed with this and stirred carefully, with the addition of not more than 150 gm. of lukewarm water. Injections of from 50 gm. to not more than 100 gm. at a time, in a lukewarm state, by means of a simple funnel, ending in a nozle which must have a wide opening. The mixture will keep only a short time. One hundred grams equal about 120 calories.

2. Nutritive Enema (*According to Ewald*).—Two or three eggs are beaten smooth with one teaspoonful of cold water and a little salt—as much as can be held on the point of a knife. Wheaten starch, as much as can be held on the point of a knife, is boiled with half a cup (100 gm.) of a 20 per cent. solution of grape-sugar and one wineglass (150 gm.) of red wine added. Then the solution is cooled to 30° R., and the eggs are stirred in slowly. One can add also one teaspoonful of meat peptone, but this is not absolutely necessary. Nutritive clysters are to be injected while at blood-heat, and in quantities of 250 gm. at a time. Previously the rectum must have been cleansed by a purgative clyster. The addition of grape-sugar had better be omitted, since through it decomposition and irritations of the intestines may arise (Wegele). It contains about 400 calories.*

3. Nutritive Clyster (*According to Boas*).—Warm 250 gm. of milk, stir with two yolks of eggs, one teaspoonful of common salt, and one tablespoonful of wheaten starch, and afterward add one tablespoonful of red wine. If the mucous membrane of the rectum is easily irritated, one may add four or five drops of tincture of opium. Such clysters may be administered from one to four times in twenty-four hours (heated to blood-heat), with a long, soft, rectal tube and a Hegar's funnel. Contains about 400 calories.

4. Meat Bouillon Wine Clyster (*According to Fleiner*).—This consists of eighty grams of beef-tea and forty grams of mild white wine; to be injected two or three times a day at body-heat. According to Fleiner, these clysters bring sleep to weak patients.

Jaccoud recommends 250 gm. of bouillon, 120 gm. of wine, yolks of two eggs, 5 to 20 gm. of peptone. *Rosenheim* uses peptone (one to two drams), two eggs, 15 gm. of glucose, and sometimes, if desired, emulsions of cod-liver oil. *Singer's* enema ("Centralblatt d. ges. Therap.," März, 1895) is very much like that of Boas, with the addition of peptone. These examples will amply suffice for all purposes.

Method and Technic of Rectal Feeding.

1. Every nutritive enema must be preceded by a cleansing injection one hour previously.
2. The amount of injected nutriment must not exceed ¼ of a liter (eight ounces) at a time.
3. After the injection the patient must remain in the recumbent position for one hour, and a hot towel should be held firmly against the anus by means of a binder for fifteen or twenty minutes.
4. The patient should lie on his left side, with his hips raised upon a pillow, and the injection must be given very slowly.

---

* In calculating the value in calories of the nutritive clysters, it is to be noted that the amount of resorption is difficult to determine, since it depends upon the state of the intestines, the skill of the patient in retaining the enema, etc. It is therefore well to assume only one-half as resorbed.

5. If the rectum is very irritable, the addition of a few drops (10 to 20) of tincture of opium is serviceable.

6. The injection should be made with a funnel or an irrigating bottle, never with a syringe. The best tube to use is that named after Langdon, as it is sufficiently soft and flexible and can not kink upon itself.

7. The tube should, in adults, be passed high up into the colon; if possible, 14 to 18 inches should be introduced, but 12 inches will, as a rule, suffice. The higher up the enema is placed, the less will be the liability of its rejection. An anatomical and physiological reason for placing injections high is found in the nature of the anastomoses of the vascular supply of the rectum, sigmoid, and colon. The superior rectal and sigmoid veins communicate with the inferior mesenteric vein; therefore these veins conduct whatever they have absorbed directly to the liver through branches of the vena porta. In the liver the very important secondary digestion takes place. The veins from the lower third of the rectum communicate with the inferior vena cava, and their contents are not conducted to the liver.

8. The temperature of the injection should be that of the body—98.6° F.

Indications Necessitating Rectal Feeding.—There are two classes of conditions in which nutritive enemata are indicated:

1. The first class comprises patients that are still able to swallow food and willing to do so, but on account of the existence of some gastric, esophageal, or intestinal disease, it is necessary or expedient to rest the stomach and bowel and exempt them from work. These are:

(*a*) *Gastric Ulcer.*—For the purpose of keeping the ulcer free from irritation and permitting it to heal or to prevent the starting up of hematemesis.

(*b*) *Dilations.*—Either in the atonic or benign forms, to attempt a cure by relieving the stomach of the weight of ingesta and the constant fermentation; or in the malignant, pyloric, and stenotic forms, because food positively can not pass the pylorus and gastro-enterostomy is refused or impossible.

(*c*) *Severe gastric irritations*, as in toxic gastritis.

(*d*) *Exhausting diseases*, especially the infectious types, when secretion and absorption are inhibited and food is not retained, though swallowed.

(*e*) *Ulcer of the esophagus or duodenum, stricture, ileus, invagination, volvulus, stenosis* of any part of the alimentary tract between stomach and rectum.

2. The conditions in which the patients are unable to swallow food are:

(*a*) Temporary obstruction to the entrance of food into the alimentary canal; presence of new growths; foreign bodies; acute inflammations about mouth, pharynx, and esophagus.

(*b*) Extreme sensitiveness of the mouth and esophagus, excited by corrosive poisons.

(*c*) Carcinoma, cicatricial contraction, diverticulum, neoplasms of esophagus, carcinoma of cardia.

(*d*) Reflex vomiting, as in pregnancy and sea-sickness.

There are *other states* in which the patients are either unable or *unwilling* to swallow food, but in these *feeding by the stomach-tube* is preferable to rectal feeding. These are: (1) Inability to swallow from coma, delirium, or paralysis of the muscles of deglutition; postdiphtheric paralysis. (2) Insanity, refusal of food. (3) Total anorexia (hysterical, etc.).

Intravascular and Hypodermic Feeding.—In 1850 Hodder first practised intravenous injection of milk in cases of collapse from Asiatic cholera. T. G. Thomas about this time published a case in which ½ of a pint of milk warmed to body-temperature was injected into one of the brachial veins, with the result of saving life. According to Gilman Thompson (*l. c.*, p. 383), Fowler has practised intravenous injection of peptone and has also given six ounces of digested beef solution in this manner. There seems to us no physiological reason why intravenous or even intra-arterial feeding should not be practised in emergencies. As a safeguard, however, we would suggest that every precaution be taken to have the injection absolutely sterile, and composed of such substances as are normal to the blood, such as serum albumin, sterile plasma, and defibrinated fresh sterile blood. Much careful experimenting is required, however, before we can be justified in using such methods on the sick human being. Intravenous and intra-arterial injections of warm sterile normal salt solutions have been extensively used in Asiatic cholera and in exhausting hemorrhages. I have used them in states of collapse in abdominal typhoid fever (typhoid intoxication) not only to raise the blood pressure, but for the purpose of flushing out the toxins when the blood pressure was still fairly good, and also had occasion to use them in hematemesis after gastric ulcer, with the conviction that life was saved thereby. Transfusion of sterile normal salt solution into the areolar connective tissue of the breast is to be preferred to these methods, because it requires only a pressure bottle, rubber tube, and sterile sharp-pointed cannula.

Subcutaneous Feeding.—In 1869 Menzel and Perco injected fats, albumin, and sugar into dogs and human beings, and showed that liquid oils were resorbed without causing local or general reaction. They injected nine grams of oil into one patient at Billroth's clinic, who had spinal caries; a swelling as large as a silver dollar ensued, but disappeared entirely in thirty hours (" Wien. med. Wochenschr.," 1869, No. 31).

Attempts have been made in the human being with injection of defibrinated calf's blood by Landenberger (" Würtemberg. med. Correspondenzbl.," Bd. XLIV, No. 20), with olive oil by Krueg (Referat in " Wien. med. Wochenschr.," 1875, No. 34), with olive oil and milk by Whittaker (" Schmidt's Jahrb.," Bd. CLXXVII, Heft 1), who in eight sittings injected 124 gm. in one day—in all he made sixty-eight injections. Karst recommended defibrinated blood (" Berlin. klin. Wochenschr.," 1873, No. 49). Eichhorn was so enthusiastic with his injections of milk, peptone, and cod-liver oil that he believed the normal nutrition of an animal could be supplanted by this method (" Wien. med. Wochenschr.," 1881, No. 32, 33, and 34). Leube proved that oils injected subcutaneously were actually used up in the metabolism of the body (Leube, " Verhandlungen des XIV. Congresses f. innere Medicin," 1895). In a case of benign hyperplastic pyloric stenosis complicated by colitis the author and his associate, Dr. Harry Adler, injected twenty-four grams of sterilized olive oil under the skin daily for three weeks. Nutritive enemata were not tolerated on account of the colitis. The oil injections, which did not cause the slightest irritation, were absorbed in from four to twelve hours, and though no analyses on metabolism could be executed, it became evident that the patient was decidedly benefited by the hypodermic use of oil.

The degree to which subcutaneous injections of sterile olive oil can supplement defective alimentation has been referred to in the preceding chapters.

CHAPTER XIII.

METHODS AND MEANS OF TREATMENT.

MINERAL SPRINGS.

Very little of a definite nature is known concerning the effect of various mineral waters upon the diseased or healthy intestine. The action of some of the most important natural waters upon the stomach, and also at the same time upon the intestines, is described in the author's work on "Diseases of the Stomach," second edition, pages 313 to 328. The classification of the most important mineral springs of the United States is there given, and I refer to that chapter for information concerning this subject. Almost every country has its own classification, based upon the chemical composition of its own springs, interpreted by the local physicians.

Dr. Albert C. Peale ("Transactions of the American Climatological Association"; also "Fourteenth Annual Report of the United States Geological Survey," Washington, 1894, p. 66) classifies the mineral springs of the United States as follows:

All mineral waters are divided primarily into two great groups—(1) nonthermal, or cold, and (2) thermal. They are then treated precisely alike with reference to their chemical ingredients.

SCHEME OF CLASSIFICATION.

Group A.—Nonthermal, or cold, springs.

Group B.—Thermal springs.

Class I.—Alkaline, ...	sulphated. muriated.
Class II.—Alkaline saline, ...	sulphated. muriated.
Class III.—Saline, ...	sulphated. muriated.
Class IV.—Chalybeate, ...	alkaline. sulphated. muriated.
Class V.—Neutral or indifferent.	

At the Thirteenth International Medical Congress, held at Paris, the members of the Congress were presented with a volume of 458 pages, containing the chemical composition, mode of action, geography, and illustrations of nearly all the mineral springs of France (" Stations Hydro-Minérales Climatériques et Maritimes de la France ").

I have personally visited a large number of the better known mineral springs in this country and in Europe, and lived for three years in Wiesbaden, Germany, the center of a district abundantly blessed with natural mineral springs ; for within a few hours' ride of Wiesbaden we find Schlangenbad, Homburg, Selters, Nauheim, Schwalbach. Carlsbad, Kissingen, Baden-Baden, Wildungen, Bilin, and a number of other German springs were also visited personally. The leading practitioners at all these resorts have different conceptions of the effects of the mineral waters on the various organs of the body and on metabolism.

It is much to be regretted that the exact effect of the mineral waters on the alimentary tract and metabolism has not been studied by competent and disinterested men, in laboratories properly equipped for the purpose. W. O. Atwater, who has contributed so much to our knowledge of the influence of food on metabolism, has every means at his disposal for studying this subject in his well-equipped laboratory at Middletown, Conn. In his digest of metabolism experiments (United States Department of Agriculture, Office of Experiment Stations, Bulletin No. 45) he gives an abstract of some work done by Höfler and Navasartianz—the former studied the effect of Krankenheiler mineral water on metabolism, and the latter the Essentuki water. Judging from the abstract, I should consider the results unreliable. Dronke and Ewald have studied the effects of the Levico arsenic-iron water on metabolism ; the patients' blood was improved in quality during the use of the water ; they did not attribute it to the better living and healthier surroundings during the experiment, but to the water. These are the only references to this subject in the article by Atwater above quoted. Let us hope that the American mineral waters, or at least the most important of them, may be studied by competent investigators in the near future. Thus far all claims and assertions of the effects of mineral waters rest upon the experiences of practitioners who practise in the neighborhood of particular springs. They are, accordingly, empirical.

Modes of Action.—In intestinal diseases mineral waters, so far as I am capable of judging personally, act in three ways: (1) By lavage of the entire gastro-intestinal tract: thereby it is freed of bacteria, toxic products, mucus, and fermentative food; (2) by stimulating or inhibiting peristalsis; (3) by favorably influencing disease of other organs, particularly the kidneys, liver, and stomach, and diseases of metabolism. Before we can dwell upon the action of natural mineral waters it is necessary to emphasize that many effects attributed to these waters can be achieved by distilled water or ordinary good hydrant water of the cities. Thus, if taken cold and in sufficiently large quantities, plain cold water will dilute the gastric secretion, which will be a benefit in cases of hyperacidity. If drunk methodically and persistently, it will relieve constipation to a certain extent, whereas plain hot water will act as a diuretic, and thereby hasten the excretion of the toxic products which usually seek their way out of the organism through the urine.

The alkaline carbonated waters of the United States (see reports referred to, and also articles by S. Baruch in Hare's "System of Therapeutics") are of use in light forms of constipation. My favorite among these is the Saratoga Vichy and Geyser; but no one must expect to cure constipation by these waters alone; the other methods of treatment mentioned in the article on constipation are indispensable.

The alkaline saline waters act very much like the alkaline carbonated. The saline waters, the main constituent of which is chlorid of sodium, increase intestinal peristalsis when taken in sufficiently large quantities, and are said to improve the appetite—a visionary claim the objective proof of which is lacking. The waters which contain sulphate of sodium and sulphate of magnesia, often called "bitter" waters, are really sources of more harm than good if they are used injudiciously. Some of the foreign waters of this type—the Hunyadi János and Franz-Josef Bitterquelle—contain so much magnesium sulphate (24 per cent.) and sodium sulphate (23 per cent.) that they ought to be distinctly contraindicated in catarrhal conditions of the intestinal canal. In many cases I have observed that the intestinal and general symptoms of chronic enteritis and colitis were aggravated by the persistent use of these various strong magnesium and sodium sulphate waters. We have in this country one of the mildest waters of this sort with which nature has endowed us. Its utility consists in its mildness. It is the magnesium spring of Bedford, Pa. In the neighborhood are also valuable calcic chalybeate springs. In chronic enteritis with constipation this water should be taken on an empty stomach before breakfast, and always warmed to about 150° F. It will not cure the enteritis, but it will improve the condition and be a more hygienic substitute for purgatives and laxatives to those patients who have abused these drugs. We have also a number of springs in Saratoga which act in an analogous manner, though they are somewhat stronger in the percentage of salts. I refer to the Congress and Hathorn waters. The warm magnesia spring, called the soda-drinking spring, which is on the golf ground near the Homestead Hotel, Hot Springs, Va., has been successfully used by my private patients who were afflicted with intestinal catarrh. It contains only thirty-two grains of salts to the gallon, and for that reason it can be recommended for lavage of the intestinal tract and kidneys. It does not contain a sufficient amount of purgative salts to do harm, even when used in excess. We have a number of springs in this country which are useful in checking, to a certain extent, the frequent discharges of diarrhea. The one with which I have had most experience is the Rockbridge Alum Spring, in Rockbridge County, Va. It has seemed to me that uncomplicated cases of diarrhea were undoubtedly benefited by this water if they observed strict diet at the same time. It is also recommended in dysentery. Personally, however, I should prefer to treat all cases of dysentery at their own home, under the treatment outlined in the article on this subject, and I consider the use of any sort of mineral water in this disease as dispensable except the indifferent waters for quenching thirst.

Indications.—The only intestinal diseases which offer any place to mineral water in the treatment are light and severe cases of chronic enteritis or colitis, chronic constipation, and diarrheas. My experience with the use of these waters in these diseases is as follows: Light and less intense attacks of these diseases can be far better managed and more readily cured at the homes of the patients and without mineral waters; chronic forms of these diseases have, in my experience, in no instance been cured by any form of balneotherapy. Nevertheless, in certain cases, particularly when there is a prominent neurasthenic element or uric acid diathesis associated with the enteritis, constipation, or diarrhea, let the patient go to Bedford, Saratoga, Rockbridge Alum Springs, or whichever spring suits the indication best, but impress him with the fundamental rule, the *sine qua non* of success—viz., the strict observance of the prescribed diet, rest or exercise, as the case may be, and sufficient sleep. The experience will be that the majority of sufferers from chronic intestinal diseases can not afford to go to the mineral springs. To satisfy their desire to test the water the various waters might be tried for a time at the home of the patient. The preceding remarks concerning the curative effects of the mineral waters apply to intestinal diseases only. There are other diseases—those of metabolism and cholelithiasis—in which certain waters are of undoubted benefit.

THE MORE IMPORTANT MEDICINAL AGENTS.

1. Purgatives and Laxatives.

The abuse of chemicals is one of the most prolific causes of intestinal diseases. It is distressing that a host of new remedies, advertised by great financial concerns, are thrust into the hands of an overcredulous medical profession before even their exact chemical composition and their effect on animals have been satisfactorily studied. Even the oldest and best-known purges are not thoroughly understood with regard to their physiological action. Before detailing the more important substances used for this purpose, I wish to premise what I have to say by the statement that with calomel, castor oil, and the magnesium salts the practitioner will be able to meet all the requirements for laxatives that may confront him in the treatment of intestinal diseases. In a great many remedies used for dyspepsias and dystrypsias it is, after all, not the substance itself which acts, but the faith which the patient has in its use or which he has in the ability of the physician. The words of the Healer of Genessaret, "Thy faith has made thee whole," contain a truth which is often overlooked in modern therapeutics.

Modes of Action.—Concerning the effect of purgatives, it is known that some of them act by producing a transudation of serum into the intestinal canal. In this way Liebig first explained the action of the neutral salts. Brieger ("Arch. f. exper. Path. u. Pharmakol.," Bd. VIII, S. 335) attributed the effects of the laxatives to an excitation of the peristalsis. The neutral salts, according to him, produced their effect by a serous or aqueous transudation. Drastic purgatives in small doses act like the laxatives, but in large doses they cause an inflammatory exudate and hypersecretion. This should be remembered before prescribing the concentrated magnesium and sodium sulphate waters in chronic enteritis, where the intestinal mucosa is already much loosened by small round-cell infiltration. Actual death of the superficial columnar epithelium can be caused in animals by solutions of these salts when they are persistently poured into the gastro-intestinal canal through a tube. The death of epithelial cells is not a postmortem effect, because it can be observed in portions of the intestines excised from the animal during life.

Castor oil acts by being partially emulsified through the bile and pancreatic juice, and it is asserted that jalap, scammony, gamboge, and elaterium act in a similar manner.

Laxatives should be used not in a stereotyped or aimless manner, but according to special indications. For instance, in acute constipation from atony and relaxation of the intestinal musculature strychnin will, in the long run, be a better purgative than any other in the pharmacopeia. It can not be emphasized too often that a proper diet is the best thing for most forms of constipation. Figs, apples, prunes, fresh rhubarb plant, and tomatoes are safer laxatives in their way than calomel. Substances which simply cause a more frequent, a fuller, but still well-formed, evacuation are called laxatives. Of these, the best and safest is castor oil; then come manna and tamarinds. A mixture of sulphur and cream of tartar mixed up with syrup of oranges and tragacanth is a harmless laxative. Similarly, compound licorice powder. When there are evidences of excessive bacterial activity and acute intestinal dystrypsia, calomel is the mainstay. When there are indications of abdominal plethora, then sulphate of magnesium and sodium, seidlitz powders, effervescent citrate of magnesia, and phosphate of soda are eligible remedies. Other substances which are useful are cascara sagrada, powdered rhubarb, senna, aloes, and the rectal administration of glycerin. It is an interesting fact that opium may, under certain conditions, act as a laxative. I have described the indications for this purpose in the article on enterospasm, and particularly in that on lead colic. I have also observed numerous cases of enterostenoses where a small dose of opium which was given to relieve the severe pain also arrested a tetanic contraction of the intestine and produced evacuation of the part of the bowel between the stenosis and the anus. If there are signs of inflammatory processes in connection with obstruction and enterostenosis, laxatives should be avoided (appendicitis, acute ileus, invaginations). It will suffice in these cases to clear out the lower bowel by enemata. Whenever acute constipation is associated with gastritis, the preference is to be given to calomel. Acute constipation without gastric involvement calls for castor oil or rhubarb preparations if medicines are needed at all. Chronic constipation not due to any stenoses or obstructions should be treated without drugs whenever possible (see special chapter), but when due to stenoses of the intestine, the use of drugs, for a time at least, is unavoidable—it prevents the stagnation of fecal matter in the suprastenotic loops. The remedies that are most available in this connection are those which do not create enterospasm or pain—viz., castor oil, sodium phosphate, cascara sagrada, magnesium citrate, compound licorice powder ; but senna, aloes, magnesium and sodium sulphate, podophyllin, and gamboge must be avoided. In old people an atony of the intestine sometimes develops when the lumen is entirely permeable. Here massage of the colon, electricity, and diet may still accomplish a great deal. But if, on account of the resistance of the patient, this treatment can not be persisted in, the use of the milder laxatives is justifiable. In younger individuals, however, habitual constipation, where there is no mechanical obstruction of the intestine, should always be cured by the methods mentioned in the chapter on Constipation, to the exclusion of drugs.

Contraindications to Laxatives.—Laxatives and purgatives should not be used in chronic enteritis with diarrhea or colitis. Boas (*l. c.*, p. 195) asserts that even mild laxatives are capable of producing a slight acute enteritis which can hardly be perceived subjectively, because it rapidly subsides. If this is true of mild laxatives, it must be much more so of strong and drastic laxatives. Both Boas (*l. c.*) and von Noorden ("Zeitschr. f. prak. Aerzte," 1898, No. 1) are of the opinion that the frequent occurrence of membranous enteritis in women is largely due to the thoughtless use of purgatives more or less unsuited to the case. They have arrived at this opinion from the fact that the marked secretion of mucus becomes arrested as soon as the intestinal peristalsis is regulated by physiological or dietetic laxatives. I have spoken of this view, with which I fully agree, in the article on membranous enteritis and mucous colic. Purgatives are, furthermore, contraindicated in chronic gastritis and gastroduodenitis. If a purge is required in these conditions, particularly when associated with jaundice, very small doses—say $\frac{1}{10}$ of a grain—of calomel, repeated every hour until the laxative effect is produced, are sufficient to accomplish every indication. There can be no doubt that any effort to move the bowels artificially is a dangerous undertaking when we are confronted with the clinical picture of any form of intestinal occlusion. Naturally, the type of ileus due to fecal obstruction (ileus stercoralis) would be amenable to this form of treatment. I have described the diagnostic features of this condition in a separate chapter, particularly the characteristics of the fecal tumors. The diagnosis is not so easy as is pictured by some clinicians, and it will be wiser to use enemata in these cases, because the fecal obstruction is usually in the colon and can be reached in this way. In appendicitis purgatives are also contraindicated. Dr. John B. Deaver is about the only one, to my knowledge, who recommends them unhesitatingly. I have failed to be impressed with his arguments in favor of this form of treatment, but I have observed ten cases in which purgatives had been used by either the patient himself or the physician prior to my seeing the case. These patients seemed to suffer more than the cases in which no purgatives were given. This may, however, be due to the fact that those cases which receive no purgatives as a rule receive opium, and often too much of it. One is as bad as the other. My experience has taught me to avoid purgatives altogether in this disease, and use just enough morphin to take the keen edge from the pain: not enough to produce somnolence or to be noticeable by any change in the pulse or respiration.

2. Antidiarrheic Medicines.

Medicinal agents to check diarrhea act by three, possibly four, methods: (1) By arresting or inhibiting the peristalsis; (2) by arresting or inhibiting hypersecretion and serous transudation from the intestinal wall; and (3) by inhibiting bacterial activity in the intestine. In all efforts at checking intestinal flux by medicines attention should first be directed toward the expediency of freeing the bowel of fermenting and stagnating contents. Frequently a laxative like castor oil or calomel is the best check on a diarrhea. (4) Possibly a fourth class of antidiarrheic remedies might be added to those already enumerated. These are the so-called protective powders, which are supposed to produce a healing of irritated areas or ulcers by covering them with an inert antiseptic substance—a very hypothetical explanation when we consider that we have a surface over twenty-five feet in length to treat, and that only in extremely rare cases do we know the location of the enteritis or ulcer.

(*a*) The most important remedies which arrest peristalsis are opium and its alkaloids, particularly morphin and codein, belladonna in small doses, and atropin.

(*b*) The astringent remedies which arrest secretion and check transudation are of two classes—(*a*) the vegetable and (*b*) the mineral astringents. Among the vegetable astringents, the most important of all are tannin and its derivatives: tannalbin, the albuminate of tannic acid, tannigen, which is diacetyl of tannic acid, and tannocol. A large number of other vegetable astringents owe their effect to tannic acid—viz., catechu, ratany, oak-bark extract, blackberry root, calumba, etc. The only mineral astringents still in use are nitrate of silver, acetate of lead, alum, and aluminium aceticotartrate.

(*c*) The antifermentative remedies are exceedingly numerous, and almost every week brings forth a new one. Among the more approved are bismuth salicylate, bismuth betanaphthol (orphol), salol, betanaphthol, benzol naphthol, nosophen, tannoform, and paraform. Bismuth subgallate is both an astringent and antifermentative, and also a mechanical covering for irritated areas. Medicines used for coating inflamed intestinal areas by an inert antiseptic covering are the bismuth combinations already referred to, and, furthermore, the phosphate and carbonate of calcium.

Methods of Administration.—The drugs referred to may be administered by the mouth, by the rectum, or some few of them hypodermically. The manner of administration will vary according to the seat of the lesion which it is desired to reach. If we desire to treat an ulcer in the duodenum or a duodenitis, the medicine is best administered by the mouth. But if we desire to treat a membranous colitis or a rectal ulcer, the drug is naturally best administered by an enema or a suppository. A good rule is to spare the stomach whenever possible, and to give whichever of these medicines is needed by rectal injection or suppositories. This rule, of course, becomes imperative whenever there is much vomiting. It is not always well to arrest a diarrhea at once, for it may be a self-help of nature to free the intestine of toxic contents. This is particularly the case in cholera nostras and Asiatic cholera. If the strength of the patient permits, the diarrhea had best be left undisturbed for a few days unless it becomes too profuse. When it comes to check diarrheas that are dependent upon transient conditions in the intestine, the use of the astringent or antiperistaltic remedies should be preceded by an evacuant in all patients whose strength justifies this treatment. There are rare cases of old and enfeebled individuals where the temporary increase of the diarrhea can not be tolerated. Furthermore, already existing diarrhea should not be increased when it is accompanied by fever and the Widal reaction turns out positive (enteric or typhoid fever).

If the patients are not too feeble and if there are none of the contraindications present that are referred to in the preceding, then a dose of opium sufficiently large to act promptly is indicated. I favor twenty drops of the deodorized tincture, or ½ of a grain of the denarcotized extract of opium. It is not necessary to combine opium with any other drug for this purpose, and it is much to be regretted that the beautiful and interesting study of the therapeutic action of drugs is completely frustrated in practice by usually combining with the one active remedy several others that have no influence, or a very insignificant one, on the process under treatment. The other constituents of opium—morphin, papaverin, codein, narcotin—by no means have the influence that opium has on pain and peristalsis of the intestine. Morphin is effective against pain, but not against the diarrhea.

In the treatment of acute enteritis it is preferable to give a few large doses of opium rather than extend the dosage indefinitely by repeating many small doses. If a rational diet is continued for about a week with this treatment and the patient placed at rest, no other medication will be required.

My experiences with the vegetable astringents and their derivatives are limited to tannin, tannalbin, and tannigen, with which all that is necessary can be accomplished. The vegetable astringents containing tannin are usually of such nauseating taste that this drawback alone prevents their use in practice. Besides this, their action is doubtful. Among the antifermentative remedies, betanaphthol bismuth and bismuth salicylate are most used. Bismuth subgallate has proved useful in some cases of diarrhea of the acute and subacute types, in which there was an idiosyncrasy against opium. The following is a useful combination in these conditions:

℞.	Calcium carbonate,	25 gm.	(375 grains)
	Calcium phosphate,	25 "	(375 grains)
	Betanaphthol bismuth,	5 "	(75 grains)
	Bismuth subgallate,	5 "	(75 grains).

M. DIRECTIONS.—An even teaspoonful three times a day.

Jaworski ("Therapeut. Monatsh.," 1892, Heft 2) has suggested an artificial carbonated mineral water for solution of calcium salts in the treatment of diarrheas.

℞.	Calcium carbonate,	2 gm.	(½ dram)
	Calcium salicylate,	2 "	(½ dram).

Dissolve in one liter carbonated water. (Mild calcium water.)

℞.	Calcium carbonate,	4 gm.	(1 dram)
	Calcium salicylate,	3 "	(45 grains).

Dissolve in one liter carbonated water. (Strong calcium water.)

The Jaworski carbonated solutions are used like mineral water—*i. e.*, six ounces of the stronger solution on an empty stomach in the morning, and the same quantity of the weaker solution three times a day, preferably warm. The great majority of cases of gastric hyperacidity run their course associated with constipation. The rare cases in which diarrhea is a symptom might be treated by this solution of Jaworski. It is not well to drink too much water when diarrhea is obstinate. The entire treatment by Jaworski's solution, however, requires only two glasses of six to eight ounces each distributed through the day.

The most available American natural mineral water for the treatment of diarrhea is the Rockbridge Alum Spring, of Rockbridge County, Va. In the chapter on the Significance of the Analysis of Gastric Contents for Diagnosis and Treatment of Intestinal Diseases I referred to the frequency with which absence of HCl secretion in the stomach (achylia gastrica) is associated with diarrhea. In these cases the HCl should never be omitted in the treatment. In the treatment of fluxes due to irritative or inflammatory conditions in the colon or rectum Boas has recommended aluminium aceticotartrate, one teaspoonful to a liter of warm water; and also bismuth subnitrate, one teaspoonful to a fourth of a liter of warm water. This bismuth, according to my experiments, when simply injected into the lower sigmoid or rectum and retained, finds its way not only through the entire colon, but even through the small intestine, and can be found in the stomach when it is washed out on the following morning (Grützner's "Antiperistalsis"). For the author's experiments on the subject see "Diseases of the Stomach," pp. 210 to 214; also in chapter on Mode of Digestion of Rectal Enemata in this volume.

Concerning the remaining remedies for the control of diarrhea, I may say briefly that the fewer of them are used, the better. The abnormal increase in the production of remedies to check diarrhea is out of all proportion to any real increase in our knowledge of their physiological action. The shameless way in which some of these remedies are advertised and the bold claims of unfailing cures should arouse the just suspicion of the practitioner. Some of the substances that are extolled in this manner are undoubtedly local, as well as systemic, poisons. This can be said particularly of arsenite of copper, which is injurious no matter how small the dose. There is no remedy for diarrhea, whether acute or chronic, which can accomplish so much toward the cure of the case as careful selection of diet, rest, and external application of heat. Prompted by the solicitations of a number of fellow-practitioners, I made personal clinical studies of some of the newer antidiarrheic remedies upon private and hospital patients, and have become convinced that they are largely unnecessary and frequently harmful. One of the essential conditions to testing a new drug in this manner is that we should exclude the synergistic action of other hygienic and dietetic methods of treatment. If we, for instance, allow the patient to go on indiscriminately with any diet he may choose to eat, the remedy can hardly be said to have had a fair chance to exert its influence. As Boas correctly says, "It is not sufficient that a remedy should produce a constipating effect." This does not include a simultaneous healing effect. A cure of enteritis can be accomplished only by long-continued rest to the intestine, giving it a sparing or preserving time by administering only such diet as milk, gelatins, and scraped beef; and if the amount of caloric requirements can not be answered this way, I have often succeeded admirably by hypodermic injections of sterilized olive oil, one ounce twice daily. I have treated some cases of gastro-enteritis with injections of sterilized oil, carried out by a trained nurse, for three months at a time, injecting occasionally 150 gm. daily to supply the deficiency in the food which was taken by the mouth. I have had but one abscess in all these innumerable injections, and this was due to distinct carelessness on the part of the nurse in failing to sterilize the oil. The results of these oil injections on metabolism are very favorable.

Contraindications to Antidiarrheic Remedies.—In chronic diarrheas of long standing opium should be avoided, because it produces a sudden checking of the discharges, whereby they accumulate, stagnate, and decompose. As mercury is said to be an etiological factor in the production of intestinal ulcers,—personally I have clinical reasons for believing in this effect of mercury under certain conditions,—calomel should not be given when there is a diarrhea in which we have reason to suspect the existence of intestinal ulcers. The differentiation between diarrhea and constipation is not always an easy matter. Its recognition sometimes necessitates the exact measurement and weighing of the amount of food ingested and the amount of feces evacuated in a fixed period—say in forty-eight hours. Constipation is a disproportion between the amount of food ingested and evacuated. We may, therefore, have constipation although the stools may be thin and frequent in number. It is, therefore, necessary to avoid antidiarrheic remedies in those diarrheas which alternate with constipation until the case has been studied a sufficiently long time to determine whether it is a true diarrhea or constipation (see chapters on these subjects). In all intestinal stenoses where the occlusion is not absolute we may have transient attacks of diarrhea. It is not wise to attempt to check the frequent discharges under such conditions. If they are associated with tenesmus, one should never omit to examine the rectum with the proctoscope for possible neoplasms or rectal ulcers.

Intestinal Tonics.

Analogous to the gastric tonics there are a number of remedies which exert, or are presumed to exert, a similar effect upon the intestine. All the bitter tinctures—gentian, calumba, quassia, calisaya—are supposed to act in this way upon the intestinal mucosa. According to Ramm ("Virchow-Hirsch Jahresbücher," 1891, S. 442), these bitter substances cause an increase in the peristalsis not only of the stomach, but also of the intestine. Boas claims to have obtained a good result with the extract of Calabar bean (Extr. fav. calabar, German Pharm., 0.05 to 10 parts glycerin, ten drops three times daily). He maintains that it is of decided use in painful flatulence, and that it has no laxative effect in small doses, and has never observed any harmful side effects. It would, therefore, seem to be an available remedy for intestinal atony. In a very large experience with this class of cases I have always found that strychnin, $\frac{1}{30}$ of a grain three times a day, or tincture of nux vomica, from ten to fifteen drops, answers every purpose of a tonic to intestinal peristalsis as far as that can be accomplished by medicinal means.

Concerning the rest of the so-called intestinal tonics, I believe that their effect is produced almost exclusively by acting upon the gastric mucosa and nervous apparatus.

Medicinal Agents for the Relief of Flatulence.

These agents have already been considered in the chapter on Meteorism and Tympanites, to which reference must be had for a more exact description. These remedies act in various manners. Some, like magnesia usta, are supposed to absorb the gas; others favor its evacuation by increase in the peristalsis. Thus, strychnin and Calabar extract are excellent against flatulency. The action of the so-called carminatives is not yet thoroughly understood, but experience has taught that they are of decided use in this distressing and frequently only symptomatological condition. The old-time method of administering them in the form of teas is still the most practical; thus, teas made from peppermint, anise seed, fennel, camomile, and thyme are sometimes followed by surprisingly prompt relief. The oils made from the same substances are also effective in some cases. When there is a simultaneous constipation, the oils may be combined with magnesia usta. It is doubtful whether powdered charcoal exerts any gas-absorbing power in the intestine, because it is known that charcoal can not accomplish this effect when it is saturated with water.

Sedatives.

The only sedatives which are used to any extent in the treatment of intestinal diseases are opium, morphin, and the alkaloids of opium, particularly codein and belladonna, with its alkaloid, atropin. While the indications for the clinical application of these narcotics are very well defined, we have no satisfactory theory explaining their action physiologically. According to Nothnagel ("Virchow's Archiv," Bd. LXXXVIII, S. 84), the effect of opium is due to excitation of the splanchnic nerves which inhibit peristalsis. According to Z. von Vamossy ("Deutsch. med. Wochenschr.," 1897, No. 29), the opium effect is brought about by paralysis of a hypothetical center in the brain, which transfers the centripetal impulses reaching the brain by way of the vagus to the centrifugal fibers innervating the peristalsis of the intestine. Still a third explanation based on experimental study is that by Jacobj ("Arch. f. exper. Path. u. Pharmakol.," Bd. XXIX, 1891) and Pohl (*ibid.*, Bd. XXIV, 1894), who attribute the sedative action of opium to an inhibition of the irritability of the intestinal wall.

The two classes of diseases in which the use of opium requires especial critical judgment are appendicitis and the occlusions. There are some medical men, especially surgeons (Murphy, Deaver, and Morris), who argue that opium in any dose or form, given in these conditions, is bad practice, because, in the first place, it obscures the signs and symptoms, so that the diagnosis can not be made, and in the second place it produces a state of pseudo-euphoria (or false improvement) which prejudices the patient against an operation that may be necessary. Against the first objection may be urged that morphin or opium may be given in such doses that they do not obtund the sensations entirely or produce somnolence. Pain must be relieved. This demand is often so imperative that refusal to comply with it frequently causes a dismissal of the physician; and as the keen edge of the pain can always be dulled by $\frac{1}{6}$ of a grain of morphin without destroying the chances of a diagnosis, I recommend that it should be given in all cases of appendicitis where there is much pain. There are other clinicians who go further than this, and speak of an opium treatment of appendicitis, giving from twenty to thirty drops of the tincture of opium three or four times a day. This treatment has never gained my confidence. I have seen it carried out by other practitioners, but was disposed against it by the remarkable depression which resulted in a number of cases. The second objection, that of the false improvement which might prejudice the patient against an operation, is invalid on the face of it. While it is the duty of the physician to relieve pain, it can not be considered his duty to withhold a means of relief in order to force upon the patient the conviction of the necessity of an operation. In occlusions opium is indicated in the first periods of reaction. Its administration should be controlled by observing the pulse and general condition of the patient. The more detailed indications for the administration of opium are given in the chapters on Appendicitis and Intestinal Occlusions. It is necessary to emphasize that opium excels all its alkaloids in the quieting effect it exerts on the intestine. It is, therefore, best to use the opium and not its alkaloids in all forms of peristaltic unrest of the intestine, in enteralgia, and in enterospasm. Here it relieves pain, arrests the stormy peristalsis, and reduces the spasm in so satisfactory a way that its place in the therapeutics of these abnormalities is a safe one.

A number of articles have recently appeared ("Münch. med. Wochenschr.," No. 48)—one each by Karl Demme, Holz, and Lüttgen—concerning the treatment of intestinal occlusions by atropin. Demme's case had stercoraceous vomiting which was not improved by lavage of the stomach. He gave $\frac{3}{64}$ of a grain of atropin hypodermically. There was no improvement. After five hours $\frac{5}{64}$ of a grain was injected; then abundant evacuations occurred. His second case also had stercoraceous vomiting. He injected $\frac{5}{64}$ of a grain of atropin, which in twenty minutes was followed by cessation of pain and vomiting, and in six hours by abundant evacuation. The cases recovered. The case of Holz recovered also; but that of Lüttgen, to whom $\frac{5}{64}$ of a grain of atropin was given hypodermically (a woman sixty-five years old with ileus), died on the following day, and it seems to me, under symptoms of atropin poisoning. I have no personal experience with this method of treatment, which was first proposed by Marcinowski ("Münch. med. Wochenschr.," No. 43). When we study the anatomical nature of some forms of occlusions, particularly the internal incarcerations and strangulations, we can not fail to become convinced that a cure by atropin is absolutely impossible. In those forms of volvulus, invagination, and obturations which are not capable of restoration to integrity by nonsurgical means, I do not understand how this drug could benefit the cases. If it is given at all, it certainly should not be given in larger than the normal doses.

INTESTINAL AUTOINTOXICATION—INTESTINAL ANTISEPTICS.

Autointoxication is a self-poisoning of the organism by (1) metabolic products, of normal or abnormal formation, which the organism forms in the fulfilment of its own vital processes; (2) by toxic products of bacterial origin produced in the intestine. A satisfactory classification of the various forms of autointoxication is not practical in the present state of our knowledge. Von Jaksch classifies them into: (1) *Retention intoxications* caused by the accumulation of physiological products of metabolism in consequence of closure or insufficiency of any one path of excretion; (2) *noso-intoxications*, caused by pathological processes which alter the normal course of metabolism in such a way as to produce harmful, in place of harmless, products. Under this group von Jaksch ("Wien. klin. Wochenschr.," 1890, No. 52) distinguishes (*a*) diseases produced by disturbances of metabolism caused spontaneously in the organism, and (*b*) diseases caused by the metabolic products of bacteria. (3) *Autointoxications caused by the effect of normal substances in large quantities*, which, however, are poisonous, or of poisonous substances that have originated from normal products. It is evident in this scheme that the autointoxications for which von Jaksch establishes a separate class are logically comprised in the second class, called *nosointoxications*. For didactic reasons Albu ("Autointoxicationen des Intestinal-Tractus," Berlin, 1895, S. 7) classifies these conditions into four groups: (1) Autointoxications caused by *loss of function of an organ*; these are generally glandular diseases with or without anatomical changes. This group comprises myxedema, the strumous cachexia, pancreatic diabetes, acute yellow atrophy of the liver, and Addison's disease, which is supposed to originate in some form of atrophy of the adrenal bodies. They are diseases, then, caused by exclusion or loss of function of those organs to which the newer physiology attributes a destructive power of toxic metabolic products originating in the organism. (2) Autointoxication by *general abnormalities of metabolism* without any evident localization. To this class belong the diseases in which the intermediate products of metabolism and the products of regressive metamorphosis reach the circulation: for instance, diabetes, gout, oxaluria. (3) Autointoxication by *retention of physiological products of metabolism* in the various organs: for instance, toxic phenomena after extensive skin burns, carbon dioxid poisoning when normal respiration is interfered with, and uremia. (4) Autointoxications caused by *overproduction of physiological and pathological products* of the organism—acetonuria, diaceturia, hydrothionemia, ammoniemia, cystinuria, and diabetic and carcinomatous coma.

The intoxications originating in the intestinal tract belong to both groups (3) and (4). They are observed in various stages of acute and chronic digestive disorders; in ileus, incarcerations, and strangulations—in fact all the obstructions; in the forms of intestinal dystrypsia or indigestion described in another chapter. To class chronic obstipation among the intestinal intoxications, as Albu does, is in my opinion an error, for I have never observed the typical symptoms of intoxications associated with uncomplicated obstipation. The effects of the intestinal autointoxications express themselves in nervous phenomena that occur in the course of chronic and acute digestive disorders. Certain cutaneous affections, —urticaria, acne, pruritus, purpura,—then dyspeptic asthma, periodic vomiting, tetany, some of the forms of anemia, cystinuria, and the toxic phenomena due to helminthiasis are ascribed to this process. It is not possible to enumerate dogmatically all the autointoxications, because, speaking strictly, acetonuria and oxaluria do not belong to this group of abnormalities.

It is already conceivable that new autointoxications will be recognized and old ones will be explained away. The direct products of abnormal carbohydrate fermentation are the following: Acetic, formic, propionic, butyric, valerianic, lactic, and succinic acid, hydrogen, carbon dioxid, and marsh-gas. Among the better known products of proteid putrefaction are ammonia, carbon dioxid, sulphureted hydrogen, methyl mercaptan, cystin, leucin, asparaginic acid, phenol, cresol, paraoxyphenyl acetic acid, phenyl propionic acid, phenyl acetic acid, indol, skatol, tyrosin, alcapton (trioxyphenyl propionic acid), acetone, the manifold ptomains, alkaloidal bodies, diamin, tetramethylenediamin (putrescin), pentamethylenediamin (cadaverin), ethylenediamin, and many toxalbumins. The organism may suffer from either retention of physiological or pathological products or the overproduction of same. It is necessary to distinguish between the terms fermentation and putrefaction, which are frequently used indiscriminately. Fermentation is applied only to decomposition of carbohydrates, and putrefaction to the breakdown of proteids, with the formation of malodorous substances.

Practically speaking, there are two kinds of autointoxication. In the first group the poisons are supposed to originate in the innermost metabolism of the cells, and, accumulating, they cause disease symptoms. Here we might class uremia, icterus (certain forms), uric acid poisoning, etc. The second group comprises those conditions in which toxins arise in cavities of the body, which, it is true, are inclosed within the body, but in reality belong to the outer world—cavities which are separated from the body machinery proper by an epithelium—for instance, the bladder, the uterus, and the lumen of the intestinal canal. That self-poisoning from such cavities is possible one can hardly doubt. The condition of intoxication resulting from decomposed food, which in its putrefaction already contains or forms poisons that are delivered up through the intestinal epithelium to the blood, is well known. The next step is that in which abnormal or deranged chemistry of digestion permits the formation of toxic substances from the ingested food. It is questionable whether all the disease symptoms that have been brought together under the term "autointoxication" are correctly classified under such a heading. For instance, the poisons generated by living or dead intestinal parasites may also invade the circulation. Surely we can not logically speak of the self-poisoning of the organism in this case. One can not fail to recognize that too much importance has been attributed to autointoxications when there is not one absolutely reliable analysis of the blood in these conditions, proving it to contain a chemical toxin, or even only to exert a toxic effect upon other animals. The great majority of human subjects that are described as being afflicted with autointoxication are characterized by neuropathic or psychopathic tendencies—women, children, and high-strung, neurasthenic men. What make this conception still more doubtful in some of the cases are the sudden onset and sudden disappearance of the symptoms.

In all well-known poisonings by chemically pure substances a certain habituation develops—for instance, in morphin, alcohol, strychnin poisoning. We should expect in autointoxication either a form of chronic poisoning or a habituation without any intoxication symptoms, but this is not what we find. We find attacks ceasing as abruptly as they began. The sudden disappearance of severe nervous disturbances in constipation which follows abundant evacuation speaks against autointoxication, for we can not assume that the entire organism will be instantaneously relieved of its absorbed poison in the moment of the evacuation. That there is a nucleus of truth in the autointoxication theory is certain, but, like all comparatively new ideas, a tendency has manifested itself to apply it too widely, and to phenomena which are explainable on the basis of other well-known physiological and pathological facts. Beaumont, for instance, could produce vertigo, pallor of the face, fainting, and obscurity of the field of vision by touching the wall of the stomach of his Canadian hunter, Alexis St. Martin, with a thermometer. No change was observable in the stomach itself while these systemic effects were produced. This can hardly be termed anything but a reflex. Such reflex phenomena occur rapidly, and promptly follow upon the irritation that evokes them. But then, if we wish to assign similar phenomena to the field of autointoxications, we must recall the difficulty and slowness with which such poisons pass the intestinal wall and liver, which can not be brought into harmony with the rapid and sudden symptoms of autointoxication. They can not pass away so promptly, and therefore can not be accused of exerting any sudden effects. In only a few instances have characteristic poisons been isolated from the urine. The toxicity of the urine and the feces of certain patients, after being injected into small animals, can hardly be a convincing proof that the patient himself is the subject of systemic intoxication by poisons in the blood. Such experiments are attended with great technical difficulties and liabilities to error. The experimental animals into which the urine of such patients is injected experience manifold deviations from the normal in their circulation. The various concentrations of the urine, its different chemical and morphological constituents, permit only of vague deductions from the effects which this urine produces on small animals. I do not wish to be considered a disbeliever in the theory, but simply to express caution with regard to accepting unreservedly the conclusions of experiments which are so liable to error. It will be wiser to wait for more objective proofs.

From the knowledge of intestinal autointoxication, the idea arose of disinfecting the bowel by so-called intestinal antiseptics. In France this idea has found wide-spread enthusiasm through the teachings of Bouchard and Dujardin-Beaumetz, but the Germans are more conservative in their estimate of the pathological importance of the phenomena described in this chapter. Albu devotes a volume to this subject, and considers it a pathological entity of the same importance as inflammation. Boas and Hans Herz consider that intestinal antisepsis rests upon a very infirm basis. There can be no doubt that the doctrine of autointoxications thus far is devoid of most every objective proof. Concerning the dietetic intestinal antiseptics, the investigations of Albu ("Deutsch. med. Wochenschr.," 1897, No. 32), Schmitz ("Zeitschr. f. physiol. Chem.," Bd. XVI), Rovighi (*ibid.*, Bd. XVI), and Winternitz (*ibid.*, Bd. XVI) have proved that milk and its products—kephyr and kumiss—produce a reduction of the total ethereal sulphates in the urine, and thus answer the requirements for antiseptic food. In France a decided revolution against the spread of the autointoxication doctrine has taken place since the publications of Bardet ("Compt. rend. de la société de Thérap.," 1895).

Treatment of Intestinal Autointoxication.—The treatment is: (1) Prophylactic; (2) dietetic; (3) hydriatic—by lavage of the stomach and intestine; (4) medicinal.

The prophylaxis includes, above all things, the avoidance of all foods easily capable of fermentation and putrefaction, particularly of foods already partially in such a condition—many vegetables, particularly when not perfectly sound, cabbage, sauer-kraut, certain very odoriferous cheeses, etc. It is probable that under certain conditions eggs may give rise to the liberation of cholin from lecithin, and perhaps also neurin (Nesbitt, "Jour. of Exper. Med.," vol. IV, p. 18). Many times we can not say on physiological or pathological reasons why a food disagrees and produces intestinal autointoxications—we know it only from experience. There are certain individual idiosyncrasies which make some food sources of danger. Some individuals can not digest strawberries, others can not digest crabs, lobsters, or oysters, without experiencing symptoms of autointoxication. In some gastric conditions the addition of alcoholic substances to the food causes an intense toxicity of the stomach-contents. I have already called attention to this fact in my work on "Diseases of the Stomach," and advised that alcohol be avoided in all forms of gastric dilation. In the "Maryland Medical Journal," July 24, 31, and August 7, 1897, I described the "Relation of Intestinal Putrefaction to Albuminuria," narrating the clinical histories of three teetotalers who were subject at times to albuminuria, the urine containing also hyaline and in one case epithelial casts. The symptoms could be prevented by lavage of the colon and stomach and adhering to a strict diet of milk and beef. In one of the cases the albuminuria could be brought on almost at will by eating a large quantity of eggs, with general neglect of the remaining diet. These cases convinced me that the kidneys could be directly injured by the excretion of poisons formed in the intestines. Notwithstanding that there is an etiological relation between intestinal autointoxication and certain clinical types of disease, complete clearness does not exist concerning their pathogenesis. We have a right to demand the chemical demonstration of one or more poisons which are capable of producing a definite group of symptoms and which can be found in the intestinal canal, as well as in the blood and excretions through the perspiration and urine. And, furthermore, the definite clinical picture must be capable of experimental production by these chemical substances.

As it is impossible chemically to disinfect the intestinal canal, the best disinfectant for the alimentary tract is its normal peristalsis; and, in the absence of this, its restoration by purgatives or its replacement by lavage. Lavage of the colon has been described so frequently in the various portions of this work (see chapters on Colitis Membranacea and Exploratory Lavage of Colon) that it is unnecessary to dwell upon it to any extent in this chapter. It is best carried out with plain water. If a disinfectant is desired, I advise chemically pure boric acid, 5 : 1000, or a saturated solution of thymol in water. Fortunately for our efforts at therapeutics, by far the greatest amount of putrefaction occurs in the colon, which is capable of being thoroughly washed out. While the small intestine is the seat of fermentations, these are not so grave in their results as are the effects of the products of putrefaction. In the chapter on the Treatment of Intestinal Occlusions I have described a method for washing out the duodenum and jejunum by duodenal intubation; though I have found this practical in the treatment of occlusion, it is not necessary in the treatment of intestinal autointoxication, where I prefer to use either calomel or castor oil to move the contents of the small intestine into the colon, and washing it out thereafter by colon lavage if necessary. The poisons that have gained access to the body find their exit also through the urine and the perspiration. It is therefore logical to stimulate the action of the kidneys and of the skin. Venesection is also of undoubted use in relieving the circulation of its burden of poisons. The body can, to a certain extent, be washed through by sterile normal salt solution, injected subcutaneously after a certain amount of poison-charged blood has been drawn by venesection. This, however, is a problem of the future. At present it is not feasible in practice. In three cases of extreme typhoid intoxication I have had good results from flushing out the system by sterile normal salt solution, injected subcutaneously. I used one liter of normal salt solution, to which I added one gram of chemically pure carbolic acid. A marked increase in diuresis resulted, the subsultus became less, and the temperature fell from 1.5° to 2° F. What the effect of the carbolic acid was in these cases I am unable to state precisely. A solution of 1 : 1000 of pure carbolic acid can not destroy the typhoid bacilli, but it may inhibit their growth. At any rate the weak solution of carbolic acid acted more favorably than the simple normal salt solutions. For light forms of intestinal autointoxication, such as run their course with an excess of indican and ethereal sulphates in the urine, a diet consisting largely of milk and boiled beef, together with attention to the evacuations, of which there should be at least two in twenty-four hours, the abundant drinking of pure warm water, plenty of fresh air, sleep, and exercise, will be sufficient.

Medicinal Intestinal Antiseptics.—From a large personal experience I have gained the impression that the so-called chemical intestinal antiseptics have a very limited range of utility. I even doubt the usefulness of adding antiseptics to the lavage water, for in my experiments on the dog's colon which was connected with the external abdominal surface by an artificial anus, whereupon the entire colon was washed, from the artificial to the true anus, with antiseptic solutions, I found it was impossible to sterilize the mucosa of the colon (Hemmeter, "Études expérimentales sur l'action motrice et digestive des intestine," XIII. Congrès International de Médicine, Paris, August, 1900). Medicines taken by the mouth accomplish still less. There is no doubt that a great many of the so-called intestinal antiseptics—for instance, salol, orphol, beta-naphthol, salicylate of bismuth, nosophen, and other remedies which have been mentioned under the heading of Antidiarrheic Substances—can produce a limited amount of disinfection. Taken in sufficient quantity to produce any extensive disinfection of the intestine they act as poisons, both upon the intestinal epithelium and the systemic circulation. There can be no doubt that the future of intestinal antisepsis will find its most effective agent for treatment in a much more careful study of the various food substances and their influence on fermentation and putrefaction. Concerning the significance of the ethereal sulphates and indican as indications of intestinal autointoxication, the reader is referred to the chapter on Examination of the Urine by Dr. Harry Adler.

The normal intestine possesses bactericidal powers which are often injured by our very efforts to combat intestinal autointoxication. For instance, R. Schütz ("Berlin. klin. Wochenschr.," 1900, No. 25) has conclusively demonstrated that even castor oil and calomel may, under certain conditions, injure the normal provisions for disinfection in the intestine (see also Chap. XVIII, this volume). A similar idea has been expressed by E. Vidal ("Revue de Chir.," October 10, 1900; abstract in "Centralbl. f. Stoffwechsel-Krankheiten," Bd. 1, S. 393). He holds that the normal intestinal wall possesses substances in its secretion which have an antitoxic action, and, furthermore, that in intestinal occlusion the fatal results are not exclusively due to the mechanical obstruction, but to a stercoremic infection due to the loss of power of the intestinal wall to exert its antitoxic effect. He prepared a glycerin extract of the pig's intestine, which he injected into rabbits after having produced an artificial intestinal occlusion. The animals were prevented from dying from the results of the occlusions by these injections. That the results of experimental intestinal occlusion produced in this way on animals can not be applied to man is evident from the experiments of Senn upon dogs, which are quoted in the chapter on Intestinal Occlusion. But E. Vidal has injected his glycerin extract of pig's intestine into two human patients who had been afflicted with long-standing intestinal occlusion. He claims that the urine became more abundant, the tongue was less dry, the respirations were deeper and slower, the pulse became firmer and regular—in fact, all the symptoms of extreme intoxication disappeared. He advises that this intestinal extract be injected before and after operations for intestinal occlusion.

MASSAGE.

Intestinal massage is used in all conditions in which the peristalsis is insufficient or in which local inflammatory indurations, such as peritonitic thickenings of long standing, are to be dispersed. Massage of the stomach has been described in the author's book on "Diseases of the Stomach," second edition, page 309.

Technics of Intestinal Massage.—This can not be considered a work in which all the details of abdominal massage are to be described and enumerated. Masseurs distinguish between intestinal "*pétrissage*," a soft kneading of the intestine, the hand of the operator gliding like a caterpillar, and "*effleurage*," meaning centripetal strokings with either the palms or the tips of the fingers of both hands. Massage can not be taught by descriptions in text-books.

It can be learned only by experience at the bedside. In practice it will be generally executed not by the practitioner, but by a special masseur or trained nurse. It is important to emphasize that all inflammatory conditions of recent date contraindicate intestinal massage of any kind. In treating certain diseases of the colon, such as colitis and membranous enteritis, a combination of massage of the colon with medicated enemata has been quite effective in my experience; for colitis with diarrhea I use the effervescent calcium solutions recommended by Jaworski, and described in the chapter on Antidiarrheic Remedies, but a saturated solution of thymol, suspensions of bismuth subnitrate, and bismuth subgallate are also serviceable. The solutions are slowly forced into the colon at body-temperature—not more than one quart at the utmost. After the patient has retained the medicated enema for ten minutes the masseur begins to tap, beat, and gently knead the colon, in a manner as described on page 311 in my work on "Diseases of the Stomach." In this way the medicine is more thoroughly brought into contact with the diseased mucosa. When constipation exists, I employ this same method, but use olive oil in which thymol has been ground up in a mortar—ten grains of thymol to the pint of olive or cotton-seed oil. Intestinal massage in appendicitis is of very little utility, except in such cases as have not been operated, and in which the tumor has lasted for three or four weeks after the disappearance of the acute symptoms, and shows little tendency toward resolution. Massage in all forms of intestinal occlusion, as well as in benign and malignant neoplasms, is positively contraindicated. In constipation due to intestinal atony massage finds a proper field of utility, especially if it is combined with proper diet and gymnastic and electrical treatment. In intestinal atony due to atrophy of the muscularis massage can not produce any permanent benefit. The same can be said of the treatment of local peritonitis by this method. It is very doubtful whether massaging can effect resorption of peritonitic exudates. In membranous enteritis and colitis it has, in my experience, been of marked utility, especially when combined with the oil enemata previously described.

The practitioner should never depend on this method of treatment alone, but combine it with other approved therapeutic means, which have been described in the various chapters of this book.

HYDRIATIC TREATMENT OF INTESTINAL DISEASES.

The treatment of intestinal diseases by water is, unfortunately, not in such a state of advanced development in America as it is in Germany. We lack physicians with abundant clinical and practical experience with this method of treatment, which always requires a properly equipped sanatorium for its perfect application.

If we wish to apply a hydriatic cure, or water treatment, we must, first of all, have a clear-cut and definite diagnosis. Nothing can be more serious in its results than to treat an obscure case of abdominal trouble by hydriatic methods before a diagnosis has been made. Next we must have a definite plan as to what objects we desire to accomplish in any particular case. After we have thus had an understanding concerning the conditions that call for this treatment, we must ask ourselves whether the objects in view *can* be accomplished. The line to be followed out then is marked by the following guide-posts: (1) Diagnosis; (2) objects to be accomplished or indications for the treatment; (3) possibility and feasibility of the treatment. The water can be applied to the body in the treatment of intestinal diseases externally and internally. The following plans may underlie the treatment: (1) Derivation of the circulation to the general cutaneous surface, to the abdominal organs, or to the extremities; (2) excitation or stimulation of depressed function of certain organs—this is known as the tonic effect of hydrotherapy; (3) to produce a depressing or a stimulating effect upon morbidly increased or decreased excitability of certain nerves and nerve centers; (4) stimulation of metabolism; (5) acceleration or retardation of the circulation, and increase or diminution of the blood supply to any parts. The relief of pain, which can frequently be accomplished by hydriatic methods alone, comes under heading (3) in the previous classification. As applied to the morbid alteration of intestinal functions, I have used this method of treatment for influencing intestinal peristalsis, for the arrest of pain, and to provoke the absorption of long-standing peritoneal exudates (appendicial tumor) when an operation could not be performed. It is doubtful whether intestinal secretion or absorption can be favorably influenced by external application of water. Hydriatic treatment can not accomplish permanent cures in intestinal diseases unless it is combined with other approved means—diet, rest, electricity, medicines, according to indication.

The following are the principal methods of external application:

1. Water Bandages or Compresses.—These may be either hot or cold, and can be applied by pads of absorbent cotton or of felt or gauze quilted in layers, and by strips of sponges or spongiopilin. In practice these applications have been superseded by the various forms of poultices or by rubber bottles which are filled with hot or cold water, as may be indicated. From personal experience I have gained the impression that hot water directly applied on cheese-cloth, gauze, or spongiopilin has a more pronounced relaxing and sedative effect than when applied in the rubber bottle, which is really not hydriatic treatment, for we are not applying water, but simply heat.

2. Douches.—These are hot, cold, or temperate. Professor H. Rieder, of Munich, has described a "precision douche" ("Zeitschr. f. diät. u. physikal. Therapie," Bd. IV, S. 363), by which water of any desired temperature can be applied under any desired pressure. Those douches of this description by means of which the faradic or galvanic current can be communicated to the douching current are especially effective in gastralgias and enteralgias. The University of Munich possesses a department for the physical methods of treatment which might serve as a model for American institutions to imitate. Douches are powerful agents in promoting intestinal peristalsis, particularly the rapidly alternating hot and cold, or Scottish, douche. But wherever there is an exaggerated condition of nervous irritability,—for instance, certain forms of agitated neurasthenia,—the douches, in my experience, act harmfully. If such conditions are associated with intestinal troubles, I prefer full baths in warm water, particularly salt water, or the Nauheim effervescent bath.* The various douches used are the following: needle, fan, rain, spray, steam, Scottish, and electric douches.

3. Baths.—The baths may be full or partial. The forms which are sometimes of utility in proctalgia are the hot sitz-baths. Cold sitz-baths are of advantage in the treatment of acute rectal inflammation associated with fever.

---

* Which can be obtained from the Triton Co., 21 Cliff St., New York. These baths have gained a reputation for the treatment of the muscular and valvular diseases of the heart, but after a thorough and prolonged trial I have become convinced that they are also valuable for stimulating metabolism and favorably influencing cases of intestinal dystrypsia due to uric acid neurasthenia.

4. The Cold or Hot Packing and Massage with Wet Cloths.—This form of hydriatic treatment can not be said to have a special effect on intestinal diseases. It influences metabolism in general. Slapping or beating the abdomen with towels wrung out in cold water acts as a stimulant to peristalsis.

Nature of the Cases Adapted to Hydriatic Treatment.—The intestinal neuroses constitute the largest class of diseases to which this treatment is applicable, then obstipation, the various forms of enteritis and colitis, and certain rectal diseases already enumerated. The so-called abdominal plethora is also favorably influenced by hydriatic management; but the intestinal occlusions, neoplasms, ulcers, and dislocations contraindicate this treatment, excepting it be directed toward the relief of pain. No definite rules can be given as regards the temperature of the baths or douches. As a rule, it can be said that acute inflammatory processes—appendicitis, proctitis, localized peritonitis—are best managed by cold applications. All other forms of longer duration and not associated with fever call for hot applications, especially when they are associated with pain. Constipation is best influenced by the cold or alternating hot and cold douche, especially if a faradic current can be imparted to the stream issuing from the douche. Patients under treatment by the various methods described in this chapter should be carefully watched if the application of water at a cold temperature is part of the plan. Many intestinal sufferers have an enfeebled circulation and low arterial tension. They accordingly do not react promptly after application of external cold. Above all things, no application of cold water should be made unless the physician has assured himself that the body of the patient is quite warm. Under no conditions should cold water be applied if the patient feels cold or chilly. This is a grave error which I have seen committed repeatedly by nurses and even physicians who call themselves "specialists" in the water treatment. If an intestinal sufferer has a weak circulation, what we desire is something to increase the action of the heart before the application of the cold water. The best means to accomplish this is moderate exercise immediately before the application. If, then, a slight perspiration is evident, this, far from being a contraindication, actually constitutes the best time for a cold shower or bath. The physiological effect of the primary stimulation and cooling and the secondary rewarming of the body after the application of the cold water are powerful stimulants to metabolism. After the application of cold, the patient should again take moderate exercise, for which walking will be sufficient, provided it is not carried to the production of perspiration. Except for the treatment of constipation and in acute inflammatory conditions, in patients with a vigorous circulation I do not sanction the application of cold to the abdomen. The temperature of the water should always be ascertained by the thermometer; the lower the temperature, the shorter the duration of application, and the prompter will the reaction set in. When a cold douche or bath has been taken, the patient should rub himself or herself off personally, because the exercise is necessary to bring on the reaction; but if the bath or application has been very hot, the patient should be rubbed off by the nurse. The one indispensable condition to success is an experienced physician or nurse in the application of these methods. When applied by inexperienced hands, it becomes a source of great danger. The various bathing institutions at the springs of the United States (Hot Springs of Virginia, Arkansas, Colorado, Saratoga, Mt. Clemens, Mich.) have trained bathing masters, who have gained their knowledge from competent medical teachers abroad. But even these should not be implicitly trusted—the physician himself should see to it that the directions are accurately carried out.

The *internal hydriatic treatment* is limited to lavage or douching of the stomach, colon, and rectum. This may accomplish a twofold purpose: (1) Cleansing of the parts which are douched, and (2) producing an increased or diminished supply of blood by the high or low temperature of the water used. Dr. Fenton B. Turck has described an apparatus which he calls the "intragastric resuscitator." It consists simply of a double stomach-tube, to one end of which is attached a soft-rubber bag. The instrument was originally devised for the purpose of reducing surgical shock and collapse (Turck, "Surgical Shock," "Jour. Amer. Med. Assoc.," June 19, 1897). The bag when collapsed fits closely around the stomach-tube and is easily introduced into the stomach. Turck's resuscitator can also be introduced into the rectum and sigmoid. He claims the following advantages for his instrument: (1) The heat is applied in a uniform and diffuse manner at much higher temperature than dare be used with water in direct contact with the mucous membrane. The temperature here used (130° to 135° F.) is in most cases necessary in order to produce the desired stimulation. (2) The exhaustion produced by passing large quantities of warm water directly into the stomach, which dilates the splanchnic vessels, is avoided, while the hot water used by the foregoing method produces stimulation of this area without exhaustion. (3) The removal of the necessary secretions and nutrient material is entirely avoided. (4) The degree of temperature, as well as the quantity of water used within the bag, is under the complete control of the operator. I have as yet not had sufficient experience with this instrument to speak upon its usefulness with authority, but the principle involved appeals to me as a practical one. High temperatures promote the nutrition of parts by increasing the blood supply. They relieve pain for the same reason. They may stimulate the heart's action, because the heart naturally beats more frequently, but perhaps not more vigorously, under high temperatures (H. Newell Martin, "Studies from Biological Laboratory," Johns Hopkins University, 1886). Extensive clinical tests may, no doubt, demonstrate that the instrument has a definite field of usefulness.

TREATMENT BY ELECTRICITY.

Electricity is of advantage in the treatment of intestinal diseases for the stimulation of peristalsis and the relief of pain. On empirical grounds we know that it can accomplish these effects, but we do not know how they are brought about. In an article on the "Effect of Induction Currents on the Animal Stomach," S. J. Meltzer ("Arch. f. Verdauungs-Krankheiten," Bd. III, S. 135) reiterates his results, published in the "New York Medical Journal," June 15, 1895, according to which percutaneous and direct faradization of the stomach and intestines of animals can produce no contraction in these parts. Only when the stomach is stimulated on the peritoneal side can a contraction be produced in the pyloric end. The fundic end of the stomach does not contract under a medium strong current (10 to 30 Ra). There is always a small portion of the left end of the stomach which can not be brought into contraction by any current, no matter how strong (O–Ra). The contraction of any part of the alimentary canal is brought about by muscular structure. As the fundic end has only a very thin layer of muscular tissue and is not so abundantly supplied with nerves as the pyloric end, it follows that it can not contract as well as the pyloric end. The muscular layer of the intestine, similarly to that of the stomach, can not be made to contract if stimulated from the surface of the mucosa. It will contract when stimulated on peritoneal surface. I have convinced myself, however, that the intestine can not be made to contract with one electrode on the outside of the abdomen and the other in the rectum. The intestinal peristalsis which Boas and Ziemssen have observed following faradic stimulation of the abdomen must be due to other, perhaps mechanical, causes. The case which Boas refers to (*l. c.*, p. 177) was one of peristaltic unrest of the intestine, in which he claims that a transient increase of the peristalsis could be produced by the electric current—he does not specify by which kind of electric current. The case which Eichhorst describes ("Handb. d. spec. Path. u. Ther.," 1890, Bd. II, S. 264) is too good in its effects to be true. A case of chronic obstipation was treated for eight days without results. One application of the faradic current, it is claimed, produced active evacuation and complete cure. The question which arises is, Would not the evacuation have taken place at any rate, and was it not due to some reflex nervous effect or the remote result of the preceding treatment? Nothnagel (*l. c.*, p. 42) and Ewald ("Berlin. Klinik," H. 105, 1897) do not attribute any marked beneficial effects to the electric treatment of intestinal diseases. The literature on the experiments of electric stimulation of the stomach and intestine is reviewed in the author's work on "Diseases of the Stomach," second edition, pages 301 to 308. Meltzer ("Arch. f. Verdauungs-Krankheiten," Bd. III, *l. c.*) makes the statement (p. 135) that one may deny every physiological influence to gastric electrization and still believe in its therapeutic effect. Possibly the meaning of this author is not correctly stated in these words, for undeniably there are physiological effects, but up to the present time we have no means of demonstrating them. We can, therefore, not deny them absolutely; we can only say that with the apparatus and methods at present at our disposal we are not capable of demonstrating the effect of electricity on the stomach and intestines, but that there is such an effect it would be premature to deny, and future investigations may yet be capable of demonstrating it. From a clinical standpoint it is not necessary to demonstrate that electricity can produce certain changes in the peristalsis, chemistry, or absorption of the intestines in order to justify its therapeutic employment. We have a right to attribute greater importance to clinical experience than to the negative results of physiological experiments. It is reasonable to presume that the metabolism and nutrition of the muscle, gland, and nerve-cells of the intestine may be influenced in some way by electricity, and that future experiments may be able to demonstrate these changes. C. F. Hodge has succeeded in demonstrating the changes in the nuclei of the cells of the spinal ganglia under the influence of electric stimulation ("Amer. Jour. of Psychology," vol. I, p. 479; vol. II, p. 376).

Electricity may be applied either with both poles on the abdomen in the form of large felt-covered electrodes, or with one pole on the abdomen and one in the rectum. The latter method is the most effective for obstipation. When the rectal electrode is used, it becomes necessary to fill the rectum with 200 c.c. of lukewarm water. Boas describes a practical electrode, the metallic portion of which consists of a platinum spiral concealed in a rubber rectal tube. The upper or external end of this instrument contains a screw for attachment of the electric wire, and also a small spigot for filling the rectum with warm water through the instrument. Boas advises increasing the strength of the faradic current until strong contractions of the abdominal muscle become visible. He has used this method in the most obstinate cases of constipation, and claims to have effected permanent cures where massage, diet, and gymnastics accomplished nothing. I have personally followed out the endorectal electrization in a large number of cases of chronic atonic obstipation, and while I consider it of great value, I do not by any means believe it to be more effective than diet and massage combined. It would serve no practical purpose to discuss the relative therapeutic effectiveness of various means of treatment for constipation: they had all of them best be employed simultaneously.

Indications and Contraindications.—The intestinal diseases in which electricity can reasonably be expected to do good are intestinal atony, atonic obstipation, and the enteralgias. As a rule, the galvanic current is most effective for pain, and the faradic current best adapted to defective peristalsis.

Combination Treatment; Apparatus Combining Massage and Electricity or Hydriatic Treatment and Electricity.—A number of instruments have been invented which, while they are means of applying massage, are moved by an electric motor (vibration massage) ("Zeitschr. f. diät. u. physikal. Therapie," Bd. IV, S. 402). The effect of this instrument, when it combines physical vibration with rapid electric stimulation, must naturally surpass that of either massage or electricity used singly. In the same way a Scottish douche will act more powerfully when its current is directed against the abdomen, charged with electricity. Dr. August Laqueur has described an electrothermic compress ("Zeitschr. f. diät. u. physikal. Therapie," Bd. IV, S. 570). The compress consists of flat, elastic, and flexible pillows and of thin wires isolated by asbestos and covered with canvas. They are connected with the strong electric current coming from an ordinary Edison lamp for house illumination. A rheostat, consisting of several Edison lamps, is inserted, and the amount of current controlled by the number of lamps entering between the source of the current and the compress. This apparatus serves to supply a modified form of dry heat, and can be applied to the epigastrium or entire abdomen for the relief of pain.

Persistent and intense enteralgias have, in my experience, yielded promptly to the combined application of external heat and a strong galvanic current. A current of from twenty-five to thirty milliamperes is sent through a plate electrode covered with spongiopilin and dipped in water as hot as can be borne. The rest of the abdomen not covered by the electrode is covered by hot towels or hot-water bags during the treatment.

PART THIRD.

INTESTINAL CLINIC.

CHAPTER XIV.

DIARRHEA.

An abnormal frequency of the evacuation of feces, thin in consistence.

Nature and Concept.

The nature and concept of diarrhea is so intimately associated with that of obstipation that it was considered practical to give the details of this subject under the heading of Obstipation, to which the reader is referred for particulars.

Etiology.

It is due mainly to two morbid factors: (1) Increased amount of water in the feces, on account of the rapid passage through the jejunum and ileum not permitting of absorption, and on account of abundant secretion and transudation from the vessels and glands; (2) an increase in the rate of peristalsis in the small or large intestine. When bile pigment can be detected in the feces, it is suggestive of diarrhea originating in the small intestine. When the diarrhea is due exclusively to disturbances in the colon, bile pigment is, as a rule, absent from the evacuations.

It has been suggested to drop entirely the clinical conception of diarrhea and to class all such symptoms with enteritis and other anatomical alterations of the intestines. This would be incorrect, for there are diarrheas that run their course without any anatomical lesions whatever. Increased peristalsis of the small intestine need not cause diarrhea if the large intestine is not involved; but increased peristalsis of the colon alone causes severe diarrhea when the small intestine is quiescent. If both small and large intestines are affected, the stools will contain more and more of undigested food the further up the small intestine is involved. Etiologically, diarrheas may be considered under three headings—viz., (1) those in which the irritations arise from the intestinal contents; (2) from the nervous system; and (3) from the blood.

Under the first group the following types may be conveniently considered: (*a*) Dyspeptic; (*b*) cathartic; (*c*) stercoral; (*d*) entozoic diarrhea.

Dyspeptic and Dystryptic Diarrhea.

Caused by certain articles of food, such as fruits, cucumbers, turnips, and sour milk. Sometimes the diarrhea depends upon the combination of certain foods—for instance, fruit and beer or sour milk and cucumbers. Spoiled articles, such as cider and sour beer, and unripe fruit are more detrimental. In some persons milk causes diarrhea, and in others constipation. The gases formed in fermenting food—CO_2, H_2S, CH_4—increase peristalsis. Catarrhal changes of the intestines do not occur in the beginning, but may develop later on. The diarrhea in this case is a curative effort, and should not be checked by constipating medicines. An interesting form of diarrhea is that due to absence of hydrochloric acid in the gastric secretion (achylia gastrica), which is amenable to treatment with dilute HCl internally (see also Chap. XVIII on Intestinal Dystrypsia).

Cathartic Diarrhea.

This type is due to the ingestion of purgative medicines that act chiefly by increasing the peristalsis. Alkaline salts act by causing an endosmosis from the blood-vessels into the intestine, and also by increase of peristalsis. The stools produced by purgatives differ from normal stools mainly in their greater percentage of water—the rapid peristalsis inhibiting absorption (see Chap. XIII on Purgatives and Laxatives).

Stercoral Diarrhea.

This form exists when alternating diarrhea and constipation constitute the main symptoms; it may even exist when there is no history of constipation, but when retention of fecal matter occurs that, by its irritating effects on the mucosa, brings about diarrhea.

The gases CO_2, CH_4, H_2S, when produced during chronic habitual constipation, may bring on an exaggerated peristalsis. In the beginning stercoral diarrhea is not associated with enteritis, which, however, supervenes when diarrhea is not treated properly or when errors of diet are continued.

Entozoic Diarrhea.

It is very probable that entozoa, certain worms, and infusoria may cause diarrhea, perhaps by mechanical irritation, or, more likely, through chemical toxins that they produce. The relation of certain amebæ and cercomonas to dysentery is considered under chapter XX. It is not definitely known whether the unicellular protozoa cause diarrhea, but the thread-worm oxyuris and trichocephalus may occasion this symptom, though not invariably (see Chap. XXXIII on Intestinal Parasites).

Nervous Diarrhea.

When the previously mentioned forms can be excluded, this type of diarrhea may be suspected, though it is occasionally difficult to distinguish from the stercoral type. Nervous diarrhea may be caused by: (1) Increased irritation of the nerves that excite peristalsis; (2) paralysis of the nerves that inhibit peristalsis; (3) increased serous (neurotic) transudation; and (4) excitation of the peripheral nervous apparatus contained in the walls of the intestine. Diarrheas that occur after intense fright, fear, or terror belong to the first and second types. They are generally acute attacks, but nervous diarrhea may also constitute a chronic disease. It is usually accompanied by other nervous phenomena, such as giddiness, vertigo, cerebral oppression and congestions, a sensation of alternating heat and cold in various parts of the body, a feeling of fear and oppression around the heart, rapid breathing, palpitation—all of which symptoms may rapidly disappear after a diarrheic passage has occurred. The number of passages may vary from two to twenty in the day, consisting of liquid intestinal contents with very little mucus. The passages are often preceded by active peristaltic unrest and accompanied by tenesmus and borborygmus. Diarrhea occurs very frequently, complicated with hysteria, neurasthenia, migraine, and diseases of the genito-urinary apparatus. The diagnosis is based upon exclusion of other forms of diarrhea or diseases of the digestive tract, absence of abnormal constituents in the stool, and presence of nervous symptoms. It may, however, occur in individuals that show no signs of nervousness or neurasthenia.

Diarrhea After Taking Cold.

This form is probably brought about by reflex vascular hyperemia of the intestinal mucosa, accompanied by increased serous transudation and mucous secretion. This reflex is intermediated through the cutaneous nerves.

Diarrhea Caused by Irritants Acting from the Blood.

Under this heading may be classed the diarrhea occurring in uremia, malaria, septicemia, croupous pneumonia, erysipelas, influenza, gout, uric acid diathesis, and cholera. It is supposed that the poison formed in these diseases is absorbed into the blood-vessels, and reaches the intestinal glands with the blood, causing an enormous increase of secretion. In this way diarrhea may occur in typhoid fever long before enteritis or intestinal ulcers have developed.

Treatment.

The treatment of diarrhea might be briefly summed up as follows: (1) Rest; (2) diet; (3) external heat; (4) castor oil or calomel; and (5) opium. If the disease has not disappeared under the systematic use of these means, it is certain that it is not a simple diarrhea, but one of the forms of enteritis or colitis or dysentery, the treatment of which is considered under their proper headings. The treatment of the dyspeptic diarrhea consists in excluding all food for twenty-four hours, and giving a saline purge, such as a tablespoonful of Epsom salts or half an ounce of castor oil, or $\frac{1}{10}$ of a grain of calomel every hour. External warm applications should be made to the abdomen, and the patient should keep very quiet. The cathartic diarrhea produced by the abuse of purgative medicines should not be treated at all, but permitted to recover spontaneously. The stercoral diarrhea should be treated like the dyspeptic form, with castor oil or calomel, though in this case an enema with one quart of water is especially beneficial. The entozoic diarrhea is best controlled by diet, rest, warmth, and colon enemata consisting of five to ten grains of nitrate of silver to one quart of water; one to two drams of quinin to the quart of water is an effective injection in diarrhea due to amebæ (see Chap. XXXIII on Intestinal Parasites). The treatment of nervous diarrhea must be to remove the cause of the fundamental neurasthenia. Hydropathic and electric management may be followed by improvement. Of medicines, the bromid of strontium in doses of fifteen grains four times a day and Fowler's solution of potassium arsenite are occasionally of value. It should be given in doses of two drops four times a day, gradually increased to twelve drops. The patient should be given directions to endeavor to withstand the desire to go to stool after the first passage in the day. By so doing he readily learns to control the number of daily evacuations.

In diarrhea caused by irritants acting from the blood, the underlying causative condition will require careful investigation, and the means of treatment will necessarily vary according to the etiology.

The diarrhea due to absence of HCl from the gastric secretion calls for large doses of this acid (thirty drops before, during, and after meals; ninety drops at each meal).

The Diet.

The best diet in diarrhea is milk; if the patient is weak, milk and whisky. For chronic diarrhea the following bill of fare is recommended:

MORNING:
- 8 ounces of chocolate boiled in milk.
- 1 soft egg.
- 1 piece of toast.

FORENOON, about 11 A.M.:
- 8 ounces of a decoction of whortleberry (2½ ounces dried berries and one pint of water); or—
- 8 ounces of soup made of toasted flour and bouillon with one egg added.
- 150 grains of scraped lean meat.
- 2 ounces of well-boiled rice.

AFTERNOON, 3 P.M.:
- 8 ounces of whortleberry decoction.

EVENING:
- 8 ounces of maltoleguminose soup with one egg.
- 5 ounces of finely minced breast-meat of boiled chicken.

During the entire day 2½ ounces of toast and eight ounces of Burgundy or whortleberry wine may be added.

It is useless to consider the endless number of drugs recommended for diarrhea, but discussion may be limited to the use of the cardinal remedy—opium. There is no remedy that checks peristalsis, inhibits excessive secretion, and reduces intestinal pain so well as opium. The denarcotized extract of opium, in doses of ½ of a grain combined with five grains of tannigen, three or four times daily, will rarely disappoint even in severe cases if used in connection with diet and a preceding dose of castor oil. The discussion of the use of intestinal disinfectants and astringents will be found in the chapters on Autointoxication; Important Medicinal Agents; Intestinal Dystrypsia; and in various chapters on other diseases affecting the intestines.

CHAPTER XV.

OBSTIPATION OR CONSTIPATION—COPROSTASIS.

Concept and Nature of Constipation.

The concept and the nature of constipation are variable. In a perfectly normal condition the human being has one evacuation in twenty-four hours. Two or three evacuations in twenty-four hours do not necessarily constitute diarrhea, nor does one evacuation in two or three days necessarily constitute constipation. The deciding factor is not so much the time intervening between the period of the evacuations, as the quantity that is expelled at these periods. Constipation is characterized either by abnormally rare evacuation or, if they occur daily, by the fact that the quantity evacuated is out of proportion to the amount of food ingested, a portion of the intestinal contents remaining behind. The accumulation of the retained feces in the lower portion of the large intestine causes intestinal distress and constitutional disturbances. The main reservoir for the fecal matter is the sigmoid colon and the upper part of the rectum. The causes of the remarkable periodicity with which the contents of this part of the small intestine are expelled are as yet unknown, but they have been ascribed to the education and habituation to which most healthy human beings subject their bowels, so that by a process of adaptation the sigmoid flexure and upper rectum propel their contents into the ampulla of the rectum once in twenty-four hours. There is a form of constipation in which the defecation occurs daily, but the evacuations are very firm and insufficient in quantity. In these cases the evacuation consists not of the ingesta of the last twenty-four hours, but of the ingesta of the day previous to the last twenty-four hours. The stool is regular, but very much delayed. There may even be constipation when the stool consists of a daily thin passage but of an insufficient amount of fecal matter. If the dejections occur less frequently than once in three days, or more frequently than twice or thrice in twenty-four hours, the deviations must be considered as pathological. Such persons generally exhibit abnormal sensations and phenomena. Retardation in the evacuations, occurring at rarer intervals than those stated, inevitably brings on a state where a physiological evacuation is the exception. The constipation here is generally the result of some anatomical or functional abnormality. The amount of the daily dejections is a variable one, and is dependent upon two factors: (1) The amount of the ingesta and (2) the intensity of bacterial vegetation of the intestine. Nothnagel and Woodward are of the opinion that a large portion of the dejecta consists not of remnants of ingesta, but of countless numbers of micro-organisms. Voigt and others have shown that fecal matter is formed in the intestine while the individual is undergoing starvation. This so-called starvation excrement is considerable in quantity. L. Hermann has observed that an intestinal loop which is doubly ligated in the dog, and from which, therefore, nothing can pass out and into which nothing can enter, nevertheless will form excremental matter consisting of the products of the intestinal mucosa, mucous epithelia, and micro-organisms. The early recognition of constipation is, therefore, not possible by comparing the daily quantity of the ingesta with the daily quantity of the dejections. The facts above stated serve to explain the comparatively large evacuation which may occur in patients who consume very little food. The irritability of the neuromuscular apparatus of the intestine varies not only in different persons, but also in the same individual at different times. The observation has been made upon soldiers that were kept on the same diet, and were approximately of the same age, that some of them had one evacuation daily, others twice daily, and still others one evacuation in forty-eight hours, and not in one case were there any evidences of constipation. Hippocrates states that his contemporaries generally had two passages during the day and one during the night. This is very probably attributable to the diet upon which the ancient Greeks subsisted, for the nature and the composition, and even preparation of the food, are important factors influencing the formation of the evacuations.

Etiology.

Constipation may be caused by abnormal changes in other organs, abnormalities in the function of the intestine, and abnormalities in the intestinal contents. Nothnagel separates the various forms into three etiologically different groups:

First Group.—Here the constipation is dependent upon certain factors which may still be considered physiological. By changing these factors the constipation may be influenced.

Second Group.—In which the constipation is a sequence of definite pathological conditions and coordinated with other symptoms as a more or less important phenomenon.

Third Group.—Here the constipation is the essential feature in the clinical picture, either representing a real or only apparent independent disease—habitual constipation.

Fleischer (*l. c.*, p. 1179) distinguishes four groups or forms of constipation:

First Group.—That in which the constipation is the result of faulty nutrition and mode of life—this seems to correspond to Nothnagel's first group.

Second Group.—This embraces such cases in which the constipation coexists with other morbid symptoms as a partial or resulting phenomenon in numerous and manifold pathological conditions—this seems to cover Nothnagel's second group.

Third Group.—In this form the constipation represents an independent disease, the primary coprostasis depending more or less upon a diminished irritability of the nerves or upon a defective development of the musculature of the intestine—this seems to correspond to Nothnagel's third group.

Fourth Group.—The fourth group of Fleischer is defined as "the constipation which is a partial or resulting phenomenon of other nervous affections, particularly of neurasthenia, but in which the symptoms of constipation are so prominent as to control the clinical picture completely and give the impression of an independent disease."

In the author's opinion, this fourth group of Fleischer's is not entitled to independent recognition: it is simply a subtype, identical with the cases classed in the second group. Leaving aside, therefore, Fleischer's fourth type of constipation, we find that the types of Nothnagel and Fleischer are identical.

Following the classification given, we will cite the causes of: (1) The constipation due to certain physiological disturbances, such as insufficient ingestion of liquids, suppression of the natural desire for stool, too much indigestible solid food, or even too much very easily digestible and nonirritating food, and want of proper physical exercise; (2) here we will describe the causes of the secondary constipation, the many organic diseases by which it may be produced, and (3) the causes of constipation as an independent disease —the so-called habitual constipation, or primary coprostasis.

1. A most frequent cause of constipation found among females is due to the voluntary suppression of the natural inclination to go to stool. Next to this, the most frequent cause is found in the abuse of purgatives, which generally brings about an exhaustion of the intestinal musculature. Inflamed hemorrhoids, rectal ulcers, anal fissures, and perineal dermatitis will lead patients to suppress the natural desire for stool in order to avoid the resulting pain; naturally, the retention of the rectal contents will make them more concentrated and hard, so that the final evacuation will cause more pain than would have resulted if these sufferers had gone to stool at the proper time.

It is an acknowledged fact that persons living in cities do not, as a rule, drink enough water. The deficiency in liquids causes a concentration of the chyme. There are not a few cases of an obstinate character that can be relieved by the proper use of good drinking-water. The constipation which follows upon profuse diarrheas is not only due to exhaustion of the musculature, but to impoverishment of the blood through loss of water. Most of the water which is ingested in such cases is generally absorbed quicker and more completely in order to replace the water deficit. Increased excretion of water through the kidneys, skin, or lungs, as well as profuse sweat after pneumonia, has been known to cause constipation by abstraction of the water from the intestinal canal. Improper dressing and tight lacing are responsible for much of this disorder among women.

Improper Food.—Whenever the food is indigestible and large masses of indigestible material remain in the intestinal canal, exhaustion of the musculature may result. The chyme or fecal matter remains too long in one and the same place, or it may even entirely obstruct the canal. The succeeding masses of ingesta accumulate in front of the obstruction, and the intestinal walls are distended by gaseous fermentation. This process may lead to a putrefactive diarrhea, but absolute suppression of these evacuations may result in a similar manner from paralytic overdistention of the bowel. A diet rich in vegetables in the beginning favors frequent evacuations, which are, as a rule, very voluminous and of soft consistence. These frequent evacuations are caused by the large indigestible residue left after the digestion of vegetables, and the organic acids which result from carbohydrate fermentation. If this unsuitable food is persisted in, constipation results from exhaustion of the musculature. Trousseau has asserted that an exclusive meat diet is constantly followed by constipation; this is quite plausible, because such a diet gives rise to no gaseous formation nor to organic acids which could excite the peristalsis. Fleischer claims to have observed constipation when the diet was very digestible and nonirritating. This is, in the author's experience, an exceptional occurrence. Individual reactions to particular foods and personal idiosyncrasies are doubtless important factors in the causation. The same kind of foods may have different effects on the intestine of different people—for instance, an exclusive milk diet generally causes constipation, but there are cases in which it may cause diarrhea. Cocoa, chocolate, strong tea, all clarets, and soups and dishes containing very large amounts of flour impede the intestinal peristalsis.

Bodily Inactivity.—Proper physical exercise very much assists in regulating the intestinal evacuation. This is due to a combination of effects brought about by exercise. In the first place, the respirations are deeper and fuller, and thereby the excursions of the diaphragm are greater, giving a more effective impulse to the intestines by its movements. There is a more energetic gaseous metabolism, and the circulation of the intestine is more actively carried on. We have seen, from the experiments quoted, that the proper intestinal circulation is one of the most important conditions to insure healthy peristalsis. This is also very much favored by the regular compression of the abdominal contents, caused by the contraction of the abdominal muscles during exercise. Nothnagel is inclined to the belief that the constipation occurring in adipose subjects is to be attributed to insufficient bodily exercise. Excessive ingestion of food and bodily inactivity are frequent causes of obesity. Constipation is not a necessary accompaniment of obesity. A rational mode of life and proper physical exercise may insure regular evacuations even though the patient be adipose.

2. It will be impossible to state in this chapter all the pathological conditions in which constipation may occur as a concomitant symptom. Among the diseases which are accompanied by constipation which interest us most are the affections of the alimentary tract itself. It is natural that stenosis of the gastro-intestinal canal should cause a form of suppression of the evacuations by preventing the passage of the contents. Constipation will therefore be observed whenever an obstruction impedes the progress of the chyme; among the causes of this obstruction the author has observed floating kidney and coloptosis. Careful physical examination of female patients suffering with constipation will reveal this cause more frequently than has hitherto been supposed. Among the gastric diseases which are accompanied by constipation are hyperacidity, anacidity, supersecretion, ulcer of the stomach, dilation, and acute and chronic gastritis. Chronic gastritis, however, is, as a rule, not accompanied by constipation. The condition of the evacuation in chronic enteritis will be thoroughly discussed in the chapter devoted to Enteritis. In Nothnagel's opinion, chronic intestinal catarrh is by no means necessarily followed by a slow peristalsis. A careful differentiation of chronic enteritis from venous hyperemia should be made in these cases. Passive hyperemia may, of course, bring about constipation, though even this does not occur regularly; at the same time, this passive congestion may predispose to the development of intestinal catarrh or enteritis, but in itself it does not represent the histological and functional changes of chronic inflammation. Obstruction to the outflow of the bile, extension of intestinal inflammation to the muscular layer following inflammations of the mucosa or serosa, and fatty degenerations of the muscular layer, claimed to occur in tuberculosis and alcoholism, must inevitably interfere with normal peristalsis. Local peritonitic adhesions and all the abnormal conditions of the lungs, heart, kidneys, and liver which effect passive congestion in the inferior vena cava and in the portal circulation, thus rendering the outflow of the blood from the intestine difficult, in the majority of cases lead to constipation.

Marked interference with the intestinal peristalsis has been observed in certain acute infectious diseases. This is undoubtedly brought about by a variety of causes, among which may be mentioned the abnormal composition of the blood, which is defective in oxygen and rich in CO_2; circulatory disturbances in the intestinal walls; intense irritation of the inhibitory nerves, due to the great lack of blood or to products of metabolism; to toxins or to the specific bacteria causing the infection. It is natural to suppose that rest in bed and the altered diet, with the increased excretion of water through the lungs and skin, also aid in the interference with normal peristalsis. Constipation accompanies certain diseases of the nervous system, such as cerebral and spinal meningitis, apoplexy, tumors, chronic hydrocephalus, certain forms of myelitis, and the neuroses, such as neurasthenia, melancholia, hysteria, and hypochondriasis. Alienists attribute much importance to the intestinal torpor accompanying depressing psychoses, and it was formerly believed that melancholia and hypochondriasis were frequently caused by constipation. Nothnagel and Bouveret, however, are of the opinion that constipation is more often the result than the cause of hypochondriasis. The psychiatrist thus attributes the psychosis to the obstipation; the gastro-enterologist attributes the obstipation to the psychosis—each in turn shifting the blame to the domain of the other. In many of these cases there is, no doubt, a "vicious circle," the melancholia through general depression, lack of exercise, and general atony favoring obstipation, and the latter in turn predisposing to the psychosis by abnormal abdominal circulation.

3. Referring to the fourth group of Fleischer's, the constipation which is partial or a resulting phenomena of other nervous affections, particularly of neurasthenia, but in which the symptoms of constipation are so prominent as to control the clinical picture completely and give the impression of a primary causative constipation, while it is really only a result, the author sees no need of establishing a separate class for these cases, because Nothnagel, Dunin, and Bouveret have emphasized that habitual coprostasis is a frequent accompaniment of general neurasthenia, and that it is a result and not a cause. The remaining symptoms of the neurasthenia may be so slight or so masked that they are completely submerged under the clinical picture of constipation. Improvement or cure of the neurasthenia relieves the constipation in these cases without special treatment directed to the latter trouble. Fleiner has expressed the

opinion that constipation accompanying neurasthenia is not due to an atonic depression of the peristalsis, but rather a spastic retardation by which the contents are held back through tonic contraction of certain portions of the intestines. If this conception is correct, it would be logical to try opium first in certain spastic constipations, and not resort to laxatives until this fails. I have known very many cases of habitual constipation in persons who gave not the slightest evidence of neurasthenia, and in whom the disease could not be considered other than as a primary constipation.

Etiologically, primary constipation as an independent disease may be divided into two classes: (1) Those due to mechanical causes, such as peritonitic adhesions of the intestines obstructing rectal valves and changes in location (enteroptosis). (2) Those cases which are due to some disturbance in the neuromuscular apparatus of the bowel.

Concerning the first of these,—the peritonitic adhesions of the intestines,—the views of Virchow have been quoted in the author's work on "Diseases of the Stomach," pages 603 to 605.

Virchow emphasizes the fact that the constipation is not caused by the peritonitic adhesions, but that the accumulation of fecal matter may give rise to the development of partial peritonitis and subsequent adhesions. The primary condition here is the accumulation of putrefying fecal matter. Kinking of the bowel by peritonitis will be much more liable to cause a pronounced stenosis than constipation.

Voetsch and Leichtenstern have called attention to the causative relation of congenital dislocations of the intestine and abnormalities in its dimensions to habitual constipation. The abnormal descent of the viscera, which has been described by Glénard under the name of enteroptosis, is, in the author's opinion, of undoubted significance in this connection. The fundamental principles of Glénard's disease have been given in the volume on "Diseases of the Stomach," page 613, and in chapter XXX of this work. The theory of Glénard is that the transverse colon is attached to the pylorus by the pylorocholic ligament. Other parts of the transverse colon are movable. This ligamentous attachment is, therefore, liable to cause a kinking, which forms an obstacle to the progress of fecal matter. The ascending and the transverse colon up to this point are, according to his view, disposed to become overdistended with fecal matter in consequence of the kinking, while that portion of the transverse colon beyond the point of fixation contracts to a hard, rigid cord (*corde colique transverse*). The criticisms of the theory of Glénard by Ewald and others have already been referred to in the chapter quoted.

Dunin calls attention to the fact that a careful analysis of very large clinical material and also a corresponding number of anatomical investigations are necessary to provide the requisite basis to this theory. Without committing myself entirely to the views of Glénard, I must state that constipation which is undoubtedly due to this and no other cause—viz., coloptosis—has been repeatedly observed by me; when, however, the other abdominal viscera become dislocated also in the course of these abnormal changes, so that evidences of nephroptosis, gastroptosis, etc., are found, I believe that constipation is caused by partial stenosis of the intestine, effected through these dislocated organs, which have become superimposed and are compressing certain portions of the bowel. The right or hepatic flexure of the colon becomes dislocated more easily than the left or splenic flexure, because the splenocolic ligament limits the descent of the left flexure. The most frequent type of coloptosis is the M-shaped form. The etiological factors in the production of coloptosis are, according to Leichtenstern, the following: (1) Incomplete descent of the cecum; (2) defective development of the muscular ligaments of the colon; (3) abnormal longitudinal increase of growth of the colon in comparison to the size of the abdominal cavity; (4) abnormal length of the mesentery. There are other rare abnormalities in which the transverse colon seems to be absent, the large intestine passing immediately from the cecum to the sigmoid flexure, or it may descend from the hepatic flexure directly down into the sigmoid flexure.

Abnormal innervation of the large intestine is now almost universally accepted as a cause of constipation. The nature of this abnormal innervation is not understood—it may be congenital or acquired. There are families in which all members of several generations suffer from constipation. In case the nervous infirmity is congenital, constipation becomes marked in the first years of life; otherwise it does not make itself felt until adolescence. In this latter case the individuals thus affected are generally able to assign some direct active cause which started their state of constipation. Nothnagel, Fleischer, and Leichtenstern state that a single interruption of the regular daily evacuation, caused by a long journey on the railroad, has given the incentive to a persistent state of constipation ; acute diarrhea, interruption of an accustomed physical exercise, error in diet, and depressing emotions may do the same thing. The increased perspiration, due to long pedestrian tours, which may cause a condensation of the intestinal contents, belongs to the exciting causes. Constipation in females has been attributed to neglect of answering a natural impulse to go to stool when detained in society or during hours of instruction. Physical exercise seems to have no influence on constipation of this type, some individuals having been known to take extensive regular exercise and nevertheless suffer from this affection.

The direct results of coprostasis are perceived as uncomfortable sensations of distention, pressure, and fullness in the abdomen, which coexist with rumbling and borborygmi in the intestine.

Intestinal colic may occur and be very intense. It is probably caused by strong tetanic contractions of the intestine above the fecal stagnation, perhaps also by excessive distention of the intestinal walls by the stagnating mass and the gaseous formation. The appetite is generally diminished, and may be absent entirely in severe cases. Very frequently one will find gastric symptoms present, such as nausea, eructation, pyrosis, and pressure after eating. As these symptoms disappear entirely when there has been a satisfactory evacuation, it is probable that they are of a reflex or nervous character. The abdomen is usually uniformly distended, but when there is a partial atony of the intestine, the meteorism is not uniform. When the abdominal walls are thin, inspection, palpation, and percussion will reveal a distention of limited portions of the intestine—most frequently of the colon.

Fleiner has attempted to decide the etiological nature of the constipation from the shape and form of the feces. The small round and globular fecal masses, similar to the excrement of the sheep ("Schafkoth"), are attributed by him to the atonic form. He also considers that fecal discharges of very small caliber, as thick as a lead-pencil or the little finger, are characteristic for the spastic type; but Nothnagel asserts that this form of passage may occur in the so-called "starvation" or "hunger evacuation," which is found in states of inanition,—after carcinomatous cachexia, for instance,—when the intestine becomes very much contracted. So much is plain: that stools of a lead-pencil form do occur independently of any general nervousness or local intestinal spasm. Neither the globular "Schafkoth" (sheep excrement) nor the lead-pencil shaped stool can be regarded as evidence of stricture or stenosis in the colon.

There may be constipation, although daily evacuations occur; in this case the quantity of the ejections is considerably less than the amount of waste food should be. In milder cases of constipation the patients are able to effect a movement after the stool has been retarded for several days, but evacuation comes on only after repeated fruitless endeavors and great exertion on the part of the patient. In severe cases artificial means, such as purgatives and injections, are requisite; sometimes these even prove ineffective, and in extreme cases the accumulated and thickened fecal matter which distends the colon must be removed by manual interference, for which purpose it is sometimes necessary to place the patient under anesthesia. Cases of extreme constipation and clinical curiosities concerning this disease can be found in the article by Leichtenstern and in the book of Illoway, and in Gould and Pyle's "Anomalies and Curiosities of Medicine." Illoway (*l. c.*, p. 42) reports a case of a woman in his own practice who had but one evacuation in a month,—every thirty days a midwife came to her house and scooped out the accumulated and hardened fecal masses from the rectum,—and she did not apparently suffer much from this retention.

The condensed hard fecal masses and abundant formation of gases irritate the intestinal mucosa mechanically and chemically; in consequence of this an abundant excretion of succus entericus ensues, and, further on, serous exudates, which liquefy a part of the condensed feces, and diarrhea becomes a prominent complication. Under these circumstances the constipation continues occasionally, even after repeated and abundant diarrheic evacuations; this is caused through retention of a large fecal impaction through the center of which a passage has been left open, also by distention of the sacculæ of the colon with fecal matter, or, finally, by the formation of diverticula of the colon in consequence of powerful and persistent expansion.

The longer the feces are retained in the intestine, the drier they become—the evacuations are hard, and sometimes seem as firm as stone; this is particularly the case if the feces have accumulated in the rectum.

It frequently occurs that the hard fecal evacuations are covered by a thin coating of mucus; this appearance has, no doubt, frequently suggested the existence of a chronic intestinal catarrh with constipation, implying that the constipation is the result of the enteritis; in reality, however, the strong production of mucus is the product of the fecal stagnation, which irritates the mucosa. Gaseous distention which extends into the small intestine has been observed.

The diagnosis of the cause of constipation is one of the most difficult tasks with which the practitioner is confronted; no matter how painstaking and laborious the analysis of the symptoms may be, the etiology is indispensable to a correct and logical treatment.

H. Illoway, in an exhaustive work on "Constipation in Adults and Children" (New York, 1897), classifies the etiology of the various types of chronic constipation under the following scheme:

(A) Chronic constipation produced by well-defined morbid processes.

(B) Chronic constipation by obstruction from foreign bodies.

(C) Chronic constipation produced by congenital malformation of a section of the large bowel, by defective development of the intestinal tract, or by dislocation of any part thereof.

(D) Chronic constipation from impairment of physiological functioning alone.

Chronic Constipation from Disease.

The morbid processes which cause chronic constipation do so in various ways:

1. By obstructing the lumen of the tube.

Cicatricial narrowing of any portion of the intestinal tract, as the result of the healing of an ulcer or after dysentery (rare).

Constriction of a section of the intestinal tract by bands—after peritoneal inflammation.

Constriction of the cecum and of the jejunum—after appendicitis, typhlitis, or perityphlitis.

Cancerous disease of the large bowel (the rectum is the part most frequently affected).

Tumors in the abdominal cavity pressing upon the bowel and occluding it.

Massive exudations of blood (hematocele) or of serum into the cellular tissue of the pelvis.

Obstruction of the rectum by a retroverted uterus.

Tumors within the rectum.

Folds of mucous membrane.

2. By impairment of the secretions poured into the intestines.

In chronic diseases of the liver, when the secretion of the bile is deficient, or perhaps altogether wanting, or when the bile secreted is not of normal character.

In diseases of the pancreas. Constipation is rather frequent in diseases of this organ, and adds greatly to the severity of the suffering.

3. By inhibition of peristalsis through the nerve centers.

In chronic diseases of the brain.

In chronic affections of the spinal cord and its envelops, and in chronic forms of insanity.

In saturnine intoxication (lead paralysis; saturnine encephalopathy).

In the paralysis after diphtheria.

4. By chronic venous congestion of the intestinal circulation.

In organic diseases of the heart.

In some chronic pulmonary affections, as asthma, emphysema, etc.

5. By voluntary abstention from stool on account of the pain it causes by reason of a diseased condition of the rectum.

In hemorrhoids.

In ulcers of the rectum.

In fissure of the anus.

In chronic inflammation of the rectum (chronic proctitis).

In abnormal irritability of the rectum (irritable rectum; hysterical rectum).

6. By changes in the mucous membrane which impair its irritability, and, if they involve a more or less extensive portion of it, render it incapable of performing its physiological function in the process of digestion.

In saturnine intoxication.

In chronic catarrh of the small intestine.

In membranous enteritis.

In atrophy of a section or sections of the intestinal mucous membrane—after catarrhs.

7. By atony of the intestinal muscles produced by morbid conditions of the stomach or of the bowels.

In atony of the stomach.

In dilation of the stomach.

As a sequence of prolonged catarrh of the large bowel.

Chronic constipation from foreign bodies.

1. Intestinal obstruction by a mass of hair.
2. Voluminous intestinal concretions.

A malformation of the intestines.

1. Abnormally developed colon.

2. Undue length or size of sigmoid flexure.
3. Diverticula of the large bowel.
4. Diaphragm in the large bowel.

Enormous congenital development of the colon.

Essential primary atrophy of the large bowel.

Congenital arrest of development of the muscular apparatus of the bowel.

Chronic constipation from impaired physiological function.

1. Perverted action.
2. Imperfect performance of physiological function.

It is only to this last subdivision of constipation that the term "habitual constipation" can be applied.

Résumé of Causes Coming to the Foreground in Modern Life.

(1) Habitual neglect to attend to the calls of nature. (2) Use of food leaving an insufficient residue, the peristalsis of the intestine requiring a stimulus of residual matter. Modern food is becoming too rich in nutritive and too poor in residual material. (This subject will be considered further under the heading of Diet for Constipation.) (3) The habit of reading while at stool. As Illoway says: "Much annoyance and useless expenditure for treatment is caused by persons who attempt to do two things at the same time—viz., to empty their bowels and to fill their heads." (4) Abstention from cold water. This can not be replaced by the use of hot drinks—tea, coffee, soups, etc., or by hot water. Many persons suffering from constipation use hot water, which, in the author's experience, increases the already existing atony, because hot water establishes a condition of local intestinal hyperemia which interferes with peristalsis; besides, hot beverages are very much more rapidly absorbed than cold ones; they, therefore, are more largely secreted through the kidneys and do not aid in diluting the intestinal contents. Cold water has a stimulating effect upon the muscular and nervous elements of the intestines, and, as it is not so rapidly absorbed, it aids in the dilution of the intestinal chyme. (5) Insufficient ingestion of fats and oils. The results of adipolysis, the fatty acids and soaps formed in the intestine, and the residue of unassimilated fat are important exciters of intestinal peristalsis. Fat also performs an important office in keeping the fecal matter soft and pliable. Many city residents take an insufficient amount of fat or none at all

in their food; this is done either from an insurmountable distaste for fat, or from false hygienic considerations, imagining, perhaps, that they can not digest fat, or laboring under the delusion that it will spoil their complexion. (6) Insufficient muscular exercise. Physical exercise is a stimulant to metabolism, favors the elimination of carbon dioxid, CO_2, and the absorption of oxygen; it is an important tonic to circulation and respiration. Indolent and confining habits must, therefore, breed torpidity of the bowels. (7) Debility of the abdominal muscles. This, in the severest type, is known under the name of pendulous belly, or "Hängebauch." (8) Excessive deposit of fat in the abdominal walls and around the intestine. (9) Defective teeth or absence of teeth. (10) Excessive mental work, worry, prolonged depressing influences, melancholia, etc. (11) Old age. (12) The abuse of medicines and injections.

Symptomatology.

We will consider the symptoms of constipation under three headings:

1. Symptoms that are directly dependent upon the constipation.
2. Consequences of constipation.
3. Disturbances of the nervous system and systemic disturbances.

1. There are few **characteristic symptoms directly dependent upon the state of constipation:** (*a*) Retardation beyond the normal period. Personally, I consider an individual constipated whenever there is no full evacuation once in sixty hours. (*b*) The excrement is of an abnormal shape and deficient in water, which causes the characteristic hardness and dryness. Patients, as a rule, are unable to have a free evacuation without the aid of purgatives.

The laity, as well as many physicians, have been guilty of exaggerating the gravity of constipation and attributing many complaints and disturbances to it which stand in no causal relation with the malady. Very frequently, in the secondary forms of constipation, the symptoms of the fundamental disease are interpreted as consequences of the coprostasis. There are persons who have only one evacuation in from three to five days, and who are, nevertheless, in perfect health.

In the great majority of cases constipation persists for a long time, and intestinal disturbances, as well as those of a systemic character, may be absent entirely. The author has had numerous patients who had been excited to anxiety and fear from hearing their malady exaggerated by laymen or even by physicians. It is very essential to discover the consequences which are due directly to the constipation and those which are concomitant symptoms and not direct consequences.

In very rare cases it has been established that the coprostasis may have grave consequences: the persistent and extreme irritation of the intestinal mucosa by hard scybala, the production of putrefactive gases, and the overdistention of the lumen by accumulated feces and gases may cause chronic enteritis, peritonitic inflammation, and even perforation peritonitis, and the motor insufficiency which is due to overdistention may increase to a paralytic condition. References have already been made to the interesting studies of Virchow ("Virchow's Archiv," vol. v), in which he demonstrates the possibility of partial peritonitis resulting from accumulation of feces.

All these serious issues are, fortunately, very rare. In the great majority of instances constipation is not a dangerous disease.

2. **Consequences of Constipation.**—In coprostasis we are confronted with the results of arrest of peristalsis, distention of the intestine with residual matter, and the necessary passive hyperemia which must result therefrom. Among the consequences of this altered circulation the following conditions may be brought about: (1) Hemorrhoids. There is no doubt that hemorrhoids can be brought on by passive stagnation in the hemorrhoidal veins. In Germany, Nothnagel, Fleischer, Ewald, and Boas agree upon this point. In this country we have a talented array of rectal specialists who have made most valuable contributions to this subject—viz., Charles B. Kelsey, "Surgery of the Rectum and Pelvis"; William Allingham and Herbert Allingham, "Diseases of the Rectum"; Joseph Matthews, "Rectum, Anus, and Sigmoid Flexure"; and Van Buren, "Lectures on Diseases of the Rectum"; the various volumes of Matthews' "Quarterly Journal of Rectal and Gastro-intestinal Diseases" may be referred to for information on the subject of hemorrhoids, and last, but not least, the work of Thomas

Charles Martin (see Chap. XXXIV on Diseases of Rectum). (2) Anal fissure and erosions of the anus. (3) Appendicitis. In 152 cases of perforation from appendicitis, Fitz found fecal masses in 47 per cent. ("Transactions of the Association of American Physicians," vol. I, 1886). Fenwick has stated that out of forty-three cases of perforation from appendicitis he had found that only in three cases there existed a definite history of constipation. The author's personal experience is at variance with that of Fenwick. In fifty-two cases of appendicitis complicated by perforation which were observed in hospital and private practice, a definite history of long-standing constipation could be ascertained in thirty-seven cases. (4) Membranous colitis and enteritis. (5) Typhlitis (stercoralis). (6) Proctitis. (7) Coproliths, enteroliths, fecal tumors, and concretions. For very interesting clinical cases concerning this result of constipation see Illoway (*l. c.*, pp. 139–150). (8) Dilations of colon, sigmoid, and rectum, which, by their pressure on the iliac veins, may cause edema of the limbs and have been known to produce varicocele and ulcerations of the bowel. (9) Hernia. (10) Diverticula. (11) Diarrhea alternating with the constipation. (12) Icterus. (13) Intestinal obstruction. (14) Autointoxication. (15) Various functional disturbances of the central nervous system. (16) Various forms of neuralgia, such as lumbago, sciatica, ovarialgia. (17) Diseases of the genital organs in women. (18) Chlorosis.

A number of these consequences—for instance, hemorrhoids, autointoxication, enteritis, and membranous colitis, etc.—will be treated in separate chapters.

Coproliths (*Fecal Concretions*).—These excrement tumors are formed mainly at the flexures of the colon, in the cecum, and in the rectum. Sometimes a number of them may occur in the same individual. So long as they have not acquired a very large size and have not existed for a long time, their recognition is not difficult: they can be compressed with the fingers and altered in shape; a gradual downward motion can be made out; but frequently they give rise to very annoying diagnostic difficulties; they have been mistaken for carcinomata and sarcomata of the liver, spleen, stomach, kidneys, ovaries, uterus, omentum, mesenteric and retroperitoneal glands, and for the pelvic bones.

The diagnosis is especially difficult when they have attained a large size and become hard as stone, show no motion whatever, and when the form is alternating. When they have caused an acute enteritis or local peritonitis, they become very sensitive to pressure and have been confounded with peritonitic exudates. The diagnosis of malignant growth is strengthened by the emaciation which is caused by the anorexia. The location, movability, resistance, sensitiveness, shape, the accompanying general symptoms of a fecal tumor, and the functional disturbances of the intestine are subject to many variations. They may have any conceivable shape. Personally, the author has observed one which had very nearly the shape of a parallelogram. They may have numerous protuberances or be perfectly smooth. The condition of the evacuations gives no definite indications, as fecal tumors have been known to exist although daily evacuations occurred. It has been repeatedly observed that coproliths which were situated in the transverse colon dragged this portion down to the symphysis, where a local peritonitis ensued which held the transverse colon in its abnormal position even after it had been evacuated. When fecal concretions have existed in the colon for a long time, they may cause manifold anatomical changes, such as colitis, stercoral ulcers, peritonitis, perityphlitis, appendicitis, gangrene, and perforation. Coproliths have also been known to cause enteroptosis, by dragging various portions of the bowel from their normal position. Kinking of the bowel, compression, and axial twists have been observed in consequence of coproliths.

Fecal Colic.—Attacks of fecal colic may ensue, accompanied by severe pain and abdominal meteorism, which may lead to fainting. This complication results from an advanced accumulation of fecal matter, and although it is somewhat relieved by the passage of gas, the symptoms do not cease until satisfactory evacuation has been produced. Unconsciousness when in combination with intestinal colic and meteorism presents difficulties of diagnosis. The colicky pains are caused by the energetic tonic peristalsis above the accumulation of excrement; the establishment of the long-standing existence of constipation and the demonstration by palpation of the presence of fecal accumulations, are deciding elements in the diagnosis. Fecal colic has, however, been observed when evacuations of the bowels have been regular.

Fecal Colic Resembling Enterostenosis.—This clinical picture may be presented after obstinate and persistent constipation, when purgatives that have been administered were insufficient to cause complete evacuation of the intestine, but produced strong contractions with severe colicky pains. As these symptoms may be accompanied by pronounced collapse and vomiting, they may simulate an attack of intestinal obstruction; they generally disappear, however, as soon as the colon is emptied by irrigation or the rectum emptied mechanically. Such conditions are seen, in the author's experience, only in very aged and weak persons, who lead a sedentary life, and in cases where there is an enforced retention of fecal matter caused by fissure of the anus or hemorrhoids; it has also been observed among the insane. Actual paralysis of the colon has been known to occur, accompanied by the severe symptoms of collapse and vomiting after very prolonged coprostasis; some of these cases are reported to have resulted fatally. Illoway (*l. c.*, p. 106) saw such profound stupor in a case of prolonged constipation due to anal fissure that it was only with great difficulty that the patient could be aroused; the stupor had lasted for over three weeks.

Coprostasic Fever ("*Kothfieber*").—Périer has called attention to constipation as a cause of fever after surgical operations; the fever is regarded as an expression of autointoxication from the intestine, because it subsided after the administration of simple purgatives. Küstner, however, has explained the fever which occurs in the puerperal state and after operations on the female genital organs as due to the resorption of small foci of pus somewhere in the pelvis, the absorption being favored by the compression of the accumulated fecal masses. It is, therefore, very doubtful whether there is such a thing as coprostasic fever in the real sense of the word. In this connection it may be asserted that constipation and the accumulation of hard feces in the pelvic intestinal loops are undeniably etiological factors of uterine displacements.

3. **Systemic Disturbances and Disturbances of the Nervous System.**—*Chlorosis.*—Sir Andrew Clark was disposed to look upon chlorosis as a copremia. Obstinate constipation and insufficient oxygenation are the constant accompaniments of chlorosis. A number of English authors have attributed the cause to the absorption of ptomains, and asserted that the disease could be cured by

the use of laxatives. A number of other complex organic bodies formed by the decomposition of albuminous substances under the influence of bacteria have been suggested as causes of chlorosis and the nervous results of constipation; this subject shall be discussed in the chapter on Autointoxication.

Autointoxication in Obstipation.—Von Pfungen has attempted to demonstrate an increase of proteid putrefaction in simple chronic constipation, but it appears that his investigations were made upon patients who suffered from other organic diseases, such as myelitis and peritonitis, and in whom the processes of metabolism must have been seriously disturbed. It is hypothetical whether uncomplicated constipation with an otherwise normal digestive function is complicated by any marked autointoxication. The author's experience, gathered from many hundreds of careful quantitative analyses of the urine in chronic constipation, convinces him that autointoxication is very rare in these cases. Bouchard has observed that the development of intestinal toxins and ptomains in the intestine depends upon the retention of liquid feces. The concentration of the feces in the colon reduces putrefaction, and the pressure of the hardened masses on the mucous membrane counteracts absorption. Intense coprostasis will reduce the rate of advance of the chyme in the small intestine, and putrefaction will already begin in the ileum. Now, as the chyme is liquid in the small intestine, the conditions for the formation of intestinal toxins are very favorable. It must not be overlooked that constipation and diarrhea may coexist within the intestine without very striking external evidences. This is particularly the case in the diarrhea caused by the irritation of large masses of hardened feces. The author has already observed, at autopsies made on cases of chronic dysentery, that large masses of hardened feces distended the cecum and transverse colon; in one case a small tortuous canal had been washed out through a fecal plug, and it was through this canal that the serous diarrheal discharges occurred. C. A. Herter and E. E. Smith ("New York Medical Journal," June 22 and 29, July 6, 13, and 20, 1895) give a number of cases of constipation, with a careful quantitative determination of the preformed and combined sulphates and their ratio. The significant features of their analyses in these cases are the high combined sulphates, the excess of indican, and that the quantity of indican was certainly greatest at times when the patient was most depressed and suffering most from constipation. It is conceivable that we may have local internal diarrheas of which there is no external manifestation whatever, and that these diarrheas in limited portions of the intestines are the causes of autointoxication. In a further research Herter has established an etiological relation between certain intestinal bacteria and autointoxication (Herter, "Brit. Med. Jour.," December 25, 1897, "Indicanuria and High Ethereal Sulphates Caused by Colon Bacillus and Proteus Vulgaris").

Partial Intestinal Atony.—This conception has been first promulgated by Federn, who defines it as a condition in which a portion of the large intestine, very limited in extent, is not capable of emptying itself of its contents. The contents adhere firmly to the intestinal wall, and perhaps in consequence of this the bowel contracts tightly around the mass; it is claimed by him that in this partial intestinal atony, contraction of the blood-vessels is reflexly brought about through intermediation of the splanchnic nerves, thus effecting rise in blood pressure. When the localized atony is treated with the faradic current, the pressure, it is claimed, diminishes and the symptoms improve. Federn claims to be able to demonstrate this condition by methodical percussion, and holds that a great many pathological phenomena are dependent upon it; among these are constipation, dyspeptic and gastric disturbances, colicky pains, neuralgia, neurasthenia, hysterical conditions, Basedow's disease, cardiac insufficiency, and asthmatic conditions.

Hypochondriasis.—It requires a neuropathic predisposition to develop hypochondriasis. Constipation *per se* can not cause hypochondriasis, or, as Virchow puts it, "In certain states of excitability (lessened power of resistance, predisposition) of the nervous system, disturbances may develop in the abdominal organs which bear the stamp of exaltation in the sensory, and depression in the motor, nerves." Since Romberg's time a great many talented clinicians have considered this subject in connection with constipation, particularly Dunin, Bouchard, Nothnagel, and Senator, but Virchow's view gives in a nutshell the most plausible modern conception.

Nervous and Cerebral. Phenomena in Individuals not Neuropathic.—These sensations consist of a feeling of pressure, heaviness, and dullness in the head, sometimes increasing to pain, giddiness, and vertigo, and occasionally a sensation of heat in the head. The dependence of these cerebral symptoms upon constipation is demonstrated by the fact that they disappear entirely after satisfactory evacuation, and recur again after several days of constipation. Herter and Smith suggest that these cerebral symptoms are due to the absorption of the aromatic products of intestinal putrefaction; Nothnagel attributed them to the absorption of ptomains; Senator suggested the possibility of resorption of hydrogen sulphid, and the consequent poisoning of the system by this gas. The asthmatic disturbances and cardiac symptoms have been attributed to the upward displacement of the diaphragm and the impediment to respiration and circulation caused thereby.

Intestinal Vertigo.—Leube has attributed symptoms similar to those mentioned in the last paragraph to vertigo caused by pressure on the terminal portions of the intestine, brought about by masses of gas or feces, or even when the finger was introduced into the rectum and irritated the intestinal wall. This condition, which he designates "Darmschwindel," he believes to be brought about by pressure on the hemorrhoidal plexus of the sympathetic system.

Diagnosis.

The simple diagnosis of constipation is, as a rule, an easy matter, but in those cases in which there are daily apparently sufficient evacuations, but, nevertheless, a gradual accumulation of excremental matter occurs in the intestine, the diagnosis may present some difficulty. As has been said, a patient may complain of diarrhea and nevertheless be constipated. In these cases distention of the colon and careful percussion and palpation will reveal the presence of stagnating masses of feces. In making the diagnosis it is well to bear in mind all the causes which have already been gone over. This will enable us to make the one all-important distinction between secondary constipation due to one of the causes already enumerated and typical habitual or idiopathic constipation. Equally important is it to distinguish between spastic and atonic conditions of the bowel, as they require diagonally opposite treatments (see H. Westphalen, "Archiv f. Verdauungs-Krankheiten," Bd. VI, H. 2; also *ibid.*, Bd. VII, H. 1, S. 28). It is no easy matter to determine whether the constipation is due to a reduced irritability of the nerves of the colon and rectum or to a defective development (atrophy) of the intestinal musculature. If the coprostasis was present from the earliest periods of childhood and has existed throughout life, defective development of the musculature is probable. But if the constipation has occurred later in life and is attributed by the patient to psychic and emotional causes, or is assigned to the wilful suppression of stool, we may be assured that an inhibition of the nervous mechanism of the colon and rectum has taken place. If idiopathic constipation is relieved or cured by treatment directed to the nervous system, without special regard for influences on the colon and rectum, it may be considered as due to neurasthenia.

A serviceable method to facilitate the diagnosis of atonic constipation is to observe the time required for any food to pass through the gastro-intestinal canal. A food should be chosen which can readily be recognized in the feces. The author has made use of raisins, eaten with skins and seeds, for this purpose. In children milk will answer if given after it has been excluded from the diet for several days. Whenever a food requires longer than thirty-six hours to pass through the alimentary canal, constipation exists. This gives an idea of the rate of peristalsis, but the quantity of food ingested must also be considered, as one evacuation in forty-eight hours may be sufficient to evacuate all the ingesta of that period.

Microscopic examination of the feces gives valuable information. Inspection of the rectum by Kelly's or Kelsey's tubes will prevent errors of diagnosis when the disease is caused by stricture or neoplasm in the sigmoid or rectum. In persistent cases the colon should always be distended, for which purpose the author prefers the use of carbon dioxid gas, generated from a siphon of Vichy or Seltzer water. This distention will in most cases reveal the seat of strictures of the colon by the difficulty with which the bowel is inflated beyond the point of stenosis. Concerning the technic of examination of the rectum and colon, reference is made to the chapter on Diseases of the Rectum, by Thomas Charles Martin, in the second volume of this work, and to the article of H. A. Kelly ("Annals of Surgery," 1895) and the text-books of Kelsey, Matthews, Cribbs, and Van Buren. In women the kidneys should always be examined for possible floating kidney. Personally, the author has used the electrodiaphane for the diagnosis of stenosis and for determining the location of the colon, and can speak favorably of this method. The examination of feces has been spoken of in a separate chapter.

As H. Westphalen (*l. c.*) justly emphasizes, spastic obstipation can be caused mainly only by two deviations from the normal : (1) The resistances to the passage of the feces have increased, (2) or the driving forces have diminished. The resistances or obstacles to the downward movement can be found in the consistency of the mass to be moved or in the condition of the soft parts which the feces must pass over. In atonic obstipation it is largely the condition of the stool which can be regarded as a cause of the delayed passage, because the excrement in this form is hard and dry, but in spastic constipation it is semisoft and sticky, but the form and shape of the fecal column is very narrow (lead-pencil thickness) in spastic obstipation.

The diagnosis of spastic obstipation requires that the physician should establish the existence of a functional neurosis, for it is this condition that brings about an increased irritability of the sensory intestinal nerves, which in turn reflexly causes a condition of tonic contraction of the musculature, thereby obstructing the progress of the intestinal contents. It would be a great mistake to presume that an individual can be affected by only one form of constipation at the same time. I have occasionally observed both forms in the same patient,—sometimes alternating, sometimes existing simultaneously,—that is, one part of the bowel was in a condition of atony, due most frequently to alimentary irregularities, while the lower rectum, especially the sphincter, was in a state of almost tetanic contraction.

Spasm of the rectal sphincters is the most easily recognizable cause of spastic obstipation. But if this can not be made out, and if the chief voluntary driving force—the abdominal muscular contraction—is intact, then there must be some abnormality in the function of the rectal musculature or of the rectal folds or valves, to which Thomas Charles Martin has attributed so important a rôle in the etiology of these conditions.

Prognosis.

In the large majority of cases the prognosis as regards life is, of course, favorable. Illoway has reported one case of death from asthenia despite apparent recovery from the constipation. James T. Goodheart ("Transactions of the Clinical Society of London," vol. XIV) reports a case of death from ileus paralyticus resulting from obstipation. Constipation of long standing may cause irreparable and anatomical changes in the bowel. Dislocations, atrophy, peritonitic adhesions, and perforation are, fortunately, rare.

The prognosis as regards recovery is, as a rule, favorable, logical treatment being followed by a resumption of the normal evacuations. The following exceptions to the rule are given by a number of authorities, and my personal experience confirms them:

1. When there is a marked dislocation of the bowel of long standing, the prognosis is doubtful.
2. When the abdominal walls are very flabby and relaxed; when the belly is pendulous, so that the pressure of the abdominal walls upon the bowel is lost—although much may be done by mechanical aids, it is, nevertheless, very doubtful whether a restoration to the normal can ever be effected.
3. In old people, where, besides atony, we have a degeneration resulting from age and from the prolonged atony. There are other factors, which will be mentioned further on, that tend to make the prognosis unfavorable.
4. When large abdominal growths interfere with the peristalsis of the intestines.

Treatment.

The most urgent indication in the treatment of constipation is, of course, the removal of the stagnating fecal masses; this can not be done in a manner that will unfailingly lead to recovery unless the diagnosis is accurately established. At the onset of the consideration of treatment reference will, therefore, be made once more to the various causes which have been enumerated and to insist upon their recognition.

The various means of treatment are classified according to their curative value in the following order: Diet, massage, electricity, gymnastics, hydrotherapy, medicines, mineral spring water, psychic or suggestive methods, and mechanical methods.

The management of constipation will differ according to whether it occurs in children, in middle age, or in senile people, and according to whether it is primary or secondary, atonic or spastic. The number of methods of treatment and of drugs that have been recommended for the relief of constipation is literally endless. It is not the intention of the author to review all the systems, formulas, and drugs of this type, and, believing in the apothegm of Boerhaave, "*Simplex sigillum veri,*" he will attempt to make the account as simple as possible for a book of this kind and state only those methods with which he has had personal experience. It is to be regretted that even at the present day the treatment of constipation is purely symptomatic and empirical, and mostly limited to the use of purgatives and enemata. This should be replaced by a logical treatment directed to the cause; it is only by a severely individualizing management that constipation can be radically cured.

The detection of the causes of constipation presents many difficulties in some cases, and occasionally it is entirely impossible. The disease may, it is true, be sometimes due to an incurable fundamental condition, such as coloptosis, defective development of the intestinal musculature, or an acquired intestinal atrophy. But even when the cause seems irremovable, when it is once correctly recognized the physician will be enabled to select a more correct treatment—at least he will be able to avoid errors; for instance, in inherited defective development of the intestinal musculature a diet may be selected which will make but slight demands upon the peristalsis of the intestine, while in other cases of constipation a kind of diet is usually serviceable which leaves considerable undigested residue, thereby stimulating the peristalsis. As the causes of the disease are extremely numerous, so must the treatment be a varied one. It is frequently brought about by a faulty method of nutrition and habits of life, and is a concomitant phenomenon of neurasthenia. For these reasons it is important to regulate and prescribe a well-adapted diet, insist upon proper habits and regularity of living, and urge the use of climatic, hydropathic, and physical methods of cure. The administration of purgatives is fore, be considered constipated if they have but one evacuation in two or three days, provided that the evacuation comes on spontaneously and is satisfactory; such cases must be observed for a time, and it is the duty of the physician to prevent them from undergoing any kind of treatment. In all cases of constipation, when they are first met with, it is wise to abstain from the use of purgatives absolutely and to give small doses of opium whenever there is pain and the diagnosis is obscure. In the author's experience, opium can do no harm in such cases, but purgatives can and frequently do.

The principal thing in the treatment is first to distinguish whether it is to be directed to constipation due to: (1) Atony; (2) to a spastic condition of the musculature; (3) to dislocations of the intestine; (4) to abnormal nervous inhibitions of the peristalsis (see causes given on p. 368).

The Diet in Constipation.—It is always important, in selecting a diet, to find out the state of the gastric digestion, and the diet should be selected, if possible, with regard to the condition of the stomach also. Hyperacidity is a gastric disease which is a frequent cause of constipation and which is frequently overlooked, and is not even stated in Illoway's complete work. Coprostasis due to excessive gastric secretion will be promptly relieved by the use of alkalis, as recommended in the author's book on "Diseases of the Stomach" (*l. c.*, p. 325). In constipation due to atony the use of foods which do not form much residual fecal matter is usually forbidden; the use of concentrated foods is also interdicted, and in place of these the author recommends foods rich in water and containing much cellulose, since these are well adapted for exciting the peristalsis. He also forbids cocoa, chocolate, milk, tea, claret, burgundy, rice, and soup made of flour; also such fruits as huckleberries; instead of wheat bread, the patient should use only Graham bread, particularly together with butter and honey; all fruits are permissible excepting persimmons. A fruit dish very much to be recommended consists of equal parts of plums and figs stewed up together with sugar and a few senna leaves. Soups may be eaten if made from bouillon, together with herbs, leaves, and roots; buttermilk is a very serviceable article, as also are kephyr (one or two days old) and cider. Beer is useful in some cases, when the intoxicating effect does not become annoying. Fresh vegetables in the form of cabbage, carrots, turnips, radishes, spinach, tomatoes, cresses, and rhubarb should enter into the dietary as frequently as possible. Sauer-kraut is an article which assists many suffering from this disease. Fats in the form of butter and rich gravies should be liberally taken; the meat should always be eaten with sauces. When there is much flatulence, the addition of spices, such as pepper, salt, caraway seed, etc., is of utility. If the constipation is due to an inflammation and enteritis, the diet can not be taken in the form of coarse vegetables; then the only vegetables that are permissible are cauliflower, spinach, tender asparagus, and perhaps mashed potatoes. Wegele asserts that very highly salted foods, such as herring and caviar, favor evacuation in some people. I have had no opportunity to become convinced of the correctness of this statement. A sufficient ingestion of cold water between meals is all-important. One pint of fresh buttermilk or kephyr should be taken twice daily. The following is suggested for a detailed diet-list:

DIET-LIST FOR CHRONIC CONSTIPATION.

MORNING:

Before breakfast, Bedford Magnesia Spring Water, ½ liter.
200 gm. of milk and cereal coffee (made of roasted wheat).
30 gm. of butter.
30 gm. of honey.
100 gm. of Graham bread.
300 gm. of buttermilk.

NOON:

200 gm. of bouillon.
200 gm. of mutton.
300 gm. of crisped cabbage.
200 gm. of plums.
300 gm. of white wine, apple cider, or beer.

AFTERNOON:

300 gm. of buttermilk.

EVENING:

150 gm. of meat.
30 gm. of butter.
300 gm. of stewed apples.

For the several meals, 250 gm. of Graham or rye bread. After evening meal, 750 gm. of beer. (For the caloric value of these see "Diseases of the Stomach," pp. 236, 237.)

Patients suffering with constipation should eat moderately. Excessive ingestion of food predisposes to constipation. The drinking of hot water early in the morning, which is more frequent than one would suppose among constipated patients, is rather inimical to regular evacuations and must be emphatically forbidden. A drink of cold fresh water about one hour before breakfast and just before going to bed is of value. Illoway (*l. c.*, pp. 378–382) gives a number of very eligible dietetic preparations, the principal constituents of which are apples; a mixture of stewed apples, prunes, raisins, and figs, sweetened with sugar and flavored with lemon-peel, is palatable and efficacious.

Massage.—In the treatment of constipation due to atony massage is the *sine qua non.* Not merely massage of the abdomen is here referred to, but also general massage. Purgative medicines and enemata can not be totally avoided in the treatment of this disease, now and then, but this treatment is only symptomatic; the main object is gradually to produce the independent healthy peristalsis of the intestine by proper manipulation. It can not be the object of this book to go into the detail of treatment by massage, which would in itself fill quite a large volume, and reference is simply made to the literature quoted. In order to be effective, this mechanical treatment must be persisted in for several months; no permanent benefit can be expected even after a treatment of one month. In the beginning of this treatment it may be necessary to use laxatives occasionally. Massage must be left to hands that are technically skilled, and must be executed correctly. Although the entire abdominal regions must be treated, the greatest importance is attributed to massage of the colon; this is executed by short pushing and beating movements, by kneading and rubbing, and even by attempting to grasp the colon between the fingers and systematically squeezing it along its anatomical course. The author has frequently been told by colleagues that the effect they expect from abdominal massage is the direct working-out of the intestinal contents; this is quite a mistake. The greatest benefit derived from systematic and skilled abdominal massage is the stimulation which the peristalsis receives and the improvement in the tonicity of the intestinal blood-vessels (see Hoffa, "Technik der Massage," Stuttgart, Encke, 1893; also Zabludowski, Z., "Massagetherapie," "Berlin. klin. Wochenschr.," 1886, 27, 28). Illoway (*l. c.*, pp. 203–263) has devoted sixty pages to this subject.

Electricity.—Most favorable results have been obtained in the treatment of constipation by competent and experienced clinicians by the faradic and galvanic currents. This treatment is efficacious in constipation due to atony, and not in the spastic form. Erb asserts that good results have been obtained by both currents in constipation due to atony, even where complete occlusion has been caused by fecal accumulations, and also in stenoses that had arisen from invagination. It has been claimed that faradization causes short contractions of the colon. The author's experiments lead him to believe that contraction of the colon can not be effected by abdominal faradization. He is convinced that this treatment is beneficial, though not nearly so much so as massage; the beneficial effect is not, however, brought about by contraction of the musculature of the colon. Leubuscher has recommended the application of the galvanic current and the introduction of one electrode into the rectum ("Centralbl. f. klin. Med.," 1887, No. 25), and Semmola has observed rapid improvement in a case of paralytic intestinal occlusion ("Bull. d. l'academ. d. méd.," 1891, September 29th and October 13th). In some cases the faradic current, in other cases the galvanic current, produces the best results. De Watteville has used both currents simultaneously—galvanofaradization (see Stintzing, Bd. v, Abth. VIII, S. 271–323). The exact way in which electricity acts is not yet understood. Personally, the author has obtained the most favorable results by a combination of both currents used at the same time (see section on Electrical Therapy).

Gymnastics—Body Movements—Swedish Movements.—That exercise will be most advantageous which produces the most frequent contractions of the abdominal muscles. But in the absence of any special direction and conduct of exercise, constipated patients are advised to follow up systematically whatever outdoor exercise is most congenial to them. Walking is a form of exercise that is within everybody's reach, but it must not be undertaken immediately after meals: at least two hours should be allowed to transpire before extensive walks are undertaken by patients. Among other exercises most to be recommended are swimming, rowing, horseback riding, and bicycle riding. All exercise should be taken in the open air, and must never be carried to exhaustion. The best guide as to whether the exercise has done good is the feeling of refreshment and invigoration, together with a stimulated appetite. Gymnastics and Swedish movements are best carried out under the guidance of an instructor; they must be kept up for several months. (Concerning methodical movements of the trunk and legs and mechanical concussion and stroking of the abdomen see Ramdohr's article in Penzoldt and Stintzing's "System," vol. V, part VII, p. 101.)

Two practical methods for training the abdominal muscles of the stomach are described in the author's work on "Diseases of the Stomach," page 623. For a very complete account of Swedish movements, massage, etc., see Illoway, page 243.

Cannon-ball Massage of the Abdomen.—Sahli recommended a three to five pound cannon-ball, covered smoothly with chamois skin or flannel; this is rolled over the course of the colon by the patient himself. The procedure is best carried out early in the morning, in bed, after the bladder has been evacuated. The knees are drawn up, and the rolling over the colon is continued for ten minutes. The most useful movements to execute with the ball are those illustrated in figure 33, page 298, of the author's work on "Diseases of the Stomach," second edition. Whenever massage by an expert or a physician is impossible, the cannon-ball method is emphatically recommended.

Hydropathic Procedures.—The external application of water may occasionally be effective in the treatment of constipation, but, as a rule, it is only an adjuvant to the methods already mentioned so far, particularly massage.

The most efficacious hydropathic method in the atonic form in the author's experience has been an alternating hot and cold douche directed against the abdomen. The water should never be colder than 20° C. This douche of alternating temperature is spoken of by some as the "Scotch douche." Sometimes, if an effect on the lower bowel is desired, the cold sitz-baths are advisable, but great caution is necessary in their application to female patients, particularly in the period preceding menstruation. Clysters, or enemata, or irrigations of the colon with warm or cold water, act mechanically, thermically, and antiseptically. The use of enemata and irrigations should be left entirely to the physician. In the spastic form H. Westphalen (*l. c.*, p. 40) highly recommends the warm alternating douches to the abdomen.

When injections are abused, the necessity for abdominal compression, which is an important element in the act of defecation, no longer exists; the abdominal muscles therefore become unaccustomed to exerting the necessary pressure, and constipation may actually become aggravated. There are three instances in which rectal and colon injections of cold water are especially indicated; these are in the constipation due to: (1) Chronic intestinal catarrh; (2) internal hemorrhoids; and (3) in that form due to icterus. Cold tub-baths, cold moist compresses on the abdomen, and cold moist rubbings are also very valuable adjuvants in the mechanical treatment of this disease, but all these methods had best be carried out in sanatoriums especially adapted to their execution.

Medicines.—Although the very best results are obtained by a systematic methodical treatment combining diet, massage, electricity, gymnastics, and hydrotherapy, the administration of purgatives can not be avoided when an advanced stagnation of feces has occurred in the intestine which can not be removed by enema. Excessive dilation of the intestinal wall by fecal accumulation seriously injures the musculature and reduces the irritability of the intestinal nerves. In view of these facts it is advisable not to wait with the administration of purgatives in these cases until the intestine empties itself by unusual exertion; such strenuous expulsive efforts are fraught with great danger to the contractile elements of the bowel. In old patients, paralytics, and those suffering from infectious fevers confining them to bed, the administration of purgatives can not be avoided. The same may be said in those patients who are, for the time being, prevented from undergoing the proper treatment; even in patients in whom dietetic, electric, and massage treatment has already effected quite an improvement, the use of purgatives is justifiable when retention of stool occurs now and then which can not be removed by an enema. On the whole, it may be said that the more rarely a purgative is used, the more effective will be its action when it is employed in an emergency. In the selection of drugs for this purpose the practitioner must carefully distinguish between atonic and spastic constipation and that due to obstruction.

When constipation is due to enteritis or colitis, the treatment will be directed to the fundamental disease; thus, in inflammation of the colon we may have constipation alternating with diarrhea, and the treatment will be regulation of diet, hydrotherapy, massage, and perhaps a few medicines. One of the few medicines in which the author has faith for constipation that has been caused by colitis is sodium phosphate in large doses. Of this he usually prescribes from thirty to sixty grains three times a day. The remaining treatment for this form of constipation will be considered under the headings of Enteritis and Colitis.

Purgatives are contraindicated when the constipation is the result of peritonitis, appendicitis, or when there is a suspicion of intestinal ulceration; furthermore, in tonic spastic contraction of the musculature, such as occurs in meningitis and lead-poisoning. In these cases the pains are increased by the purgatives and they do not produce an evacuation. Spastic contractions of limited portions of the intestines may occur as a result of neurasthenia. Here, as well as in recent hemorrhage from the stomach and bowel, purgatives must be prohibited. All stenoses of the intestine when they have reached an advanced degree are made worse by purgatives, because they favor the development of paralysis. During long-continued use of one and the same purgative its effectiveness is lost; it is therefore necessary to change them when they must be used for a long time. The conditions which are contraindications to the use of purgatives are considered in a special chapter.

The secretory, motor, and absorptive power of the stomach should be examined, and any defects corrected; many forms of constipation are caused by organic or functional lesions in the rectum; in persistent constipation, therefore, the rectum should always be examined. C. Cleveland has reported favorable results in constipation treated by forcible dilation of the sphincter ani muscle ("New York Medical Record," March, 1889). Fissure of the anus and hemorrhoids should receive proper attention. When there has been gastric hyperacidity, the excess of acid should be neutralized by alkalis, and when the acid has been found absent, it should be supplied in large doses.

Of the large number of drugs used for this disease in its atonic form, the author's faith has been placed mainly in nux vomica, with its alkaloid, strychnin, aloes, and cascara sagrada, and after years of repeated tests and careful clinical observation he has discontinued the use of physostigmatis faba, ergot, and belladonna for this purpose. When there is an evident decrease of mucus as a result of atrophy of the glandular layers of the intestine, the action of the glands may be stimulated by sodium phosphate and ammonium chlorid. Belladonna seems to have some good effect in those cases of constipation which are due to excessive hyperacidity, but its effect is very transient and it can not be given frequently.

The following formula is recommended in this form of constipation :

℞. Magnesiæ ustæ (calcined magnesia), ... gr. ccxxxiss (15.0 gm.)
Pulvis rad. rhei (powdered rhubarb), . . . ʒ iiss (10.0 gm.)
Natri carbon. (sodium carbonate), ... gr. lxx$\frac{1}{5}$ (5.0 gm.)
Ext. belladonna,
Ext. strychnin, of each, ... gr. i$\frac{7}{10}$ (0.1–0.2 gm.).
M. SIG.—One-half teaspoonful three times daily, half an hour after meals.

When atonic constipation is associated with absence of HCl or subacidity, the following is recommended :

℞. Acid. hydrochlor. dilut. (dilute hydrochloric acid), ℥ ss
Strychnin sulphate, ... gr. ⅓
Tinctura rhei (tincture of rhubarb), . . . q.s. ad f℥ vj.
M. SIG.—One tablespoonful in a wineglassful of water three times a day, one hour after meals, through a glass tube.

Each group of the purgatives has an individual and peculiar action, and should be used according to special indications only ; thus, when constipation is connected with acute indigestion and much intestinal sepsis, calomel in doses of half a grain repeated every two hours has no superior ; in acute constipation without sepsis or pain castor oil is a most reliable and effective purge. In constipation of the adipose, with inclination toward passive congestion in the intestinal canal and hemorrhoids, the neutral salts, such as magnesium and sodium sulphate, sodium and potassium tartrate, sodium phosphate, and the purgative bitter waters have gained a reputation.

The following is a favorite pill with Nothnagel :

℞. Podophyllin, ... gr. ivss (0.3 gm.)
Ext. aloes,
Ext. rhei (rhubarb), ... āā gr. xlviss (3.0 gm.)
Ext. taraxac., q.s.
SIG.—Mix and make forty pills. One pill at bedtime.

For constipation with flatulence the following pill is recommended:

℞.	Ext. colocyn. comp.,	gr. ⅓
	Terebinth. veneta (Venetian turpentine),	gr. j
	Pulv. aloes socot. (powdered Socotrine aloes),	gr. iss
	Ext. nuc. vomic.,	gr. ¼
	Ext. hyoscyam. (English),	gr. j.

SIG.—Make one pill. One pill two or three times a day.

With some persons the following electuarium of manna, as recommended by Strumph, will for a long time be serviceable:

℞.	Manna,	
	Sugar,	
	Fennel-water, of each,	30 grains
	Powdered orris root,	2 "
	Oil of sweet almonds,	15 drops.

DOSE.—A heaping teaspoonful.

The confection of scammony or of tamarinds, prepared according to the German or British pharmacopeia, is sufficient with some cases. Senna and colocynth can be used only occasionally. The saline purgatives, sodium and magnesium sulphate, and the various purgative bitter mineral waters, such as the Hunyadi János, Friedrichshaller, Homburg, Carlsbad, etc., in Germany, the Hathorn, Congress, and Kissingen spring in Saratoga, N. Y., and the Estill and Irvine springs in Kentucky should not be used habitually.

The author is convinced that the very drastic purgative waters, such as the Hunyadi János and the Rubinat-Condal, do more harm than good.

The Use of Mineral Spring Waters.—For a fuller account reference should be made to the special chapter devoted to this subject, and also to "Diseases of the Stomach," by the author, pages 301 to 315. The author has failed to observe a single case of atonic constipation that was cured by mineral spring water. These waters do not act curatively, but simply do what all other purgatives do—produce an evacuation for that day, and do not affect the underlying causative conditions of the constipation. In some cases, where the constipation is attributable to an enteritis or colitis, it may be permanently benefited by a sojourn at one or other of the mineral springs. The most important factor in the treatment of constipation—namely, the selection of proper diet and a strict observance of the same—is very much neglected at the spring resorts. The improvement which follows a so-called mineral spring cure is attributable to other causes as well as to the water ("Diseases of the Stomach," *l. c.*). The author has had considerable personal experience with the Bedford mineral springs, and in his opinion it is one of the few waters which can be taken for a long time without harm. This is due to the fact that it contains only ten grains of magnesium sulphate to the pint, being, therefore, a mild purgative, whereas the Friedsrichshall water contains thirty-nine grains to the pint, and the Pullna, in Bohemia, contains ninety-three grains to the pint, in addition to 124 grains of sodium sulphate. Unfortunately, most of these patients labor under the delusion that the more drastic a purgative the water is, the greater will be the benefit derived from its use. Exactly the reverse may be said to be the case.

Injections.—These consist of water, to which various medicines are added, of oil, or of glycerin. Injections of water, to be effective, should always be slightly cooler than the temperature of the body—about 75° F. will answer. When there is much putrefaction of the colon, it is well to order the injections every day. They are best administered before breakfast in the morning or before retiring at night. The quantity to begin with should not exceed one liter (one quart). It is sometimes advisable to follow the suggestion of Hühnerfauth and produce an evacuation of fecal matter contained in the sigmoid and rectum by injecting one pint of water, and then follow it by a larger injection of about two pints of water, persuading the patient to retain it as long as possible. In excessive putrefaction the author is in the habit of using disinfectants in the colon irrigation; the following are those recommended: Thymol, five parts to the 1000 of water; lysol, one part to 1000 of water; boric acid, twenty to thirty parts to 1000 of water.

Oil Injections.—Only the very best olive oil or cotton-seed oil is serviceable for this purpose.* The oil of the poppy- and rape-seed is also used in Germany; about from 250 to 500 c.c. of this oil are warmed by placing the bottle in a vessel containing hot water.

---

* Most of the imported olive oil sold in this country is nothing but American cottonseed oil bought by the French and Spanish and Italian exporters.

The rectal or Langdon colon tube is introduced into the sigmoid flexure, while the patient is placed either on the back, with the pelvis elevated by several noncompressible pillows, or in the knee-elbow position. A single injection of 250 c.c. of oil will not reach the cecum; the injections will have to be repeated daily if any benefit is to be derived from them. When the entire colon becomes coated with oil, the stools resemble those of the contents of the small intestine. In case abnormal putrefactive processes are to be combated, the addition of the same antiseptic substances and in the same quantity as stated above for the water injections is recommended. In the author's opinion, one of the most desirable effects of the oil injection—somewhat overlooked hitherto—is that the oil inhibits the resorption of toxins from the intestinal contents. When the bowels have been brought to act several times daily, the number of injections may be reduced to one in every two or three days. The oil enemata should always be combined with cannon-ball massage.

Glycerin injections have been recently replaced by suppositories containing about 80 per cent. of glycerin; they can be used only occasionally; sometimes, by their great dehydrating effect, they act as irritants upon the rectal mucous membrane.

Mechanical or Instrumental Methods.—When the rectum has become enormously dilated by hard impacted masses of fecal matter, it sometimes becomes necessary to remove them by manual or instrumental interference. Fecal colic caused by coproliths has been relieved by manual kneading and breaking up of the fecal tumors, when injections of water and oil did not suffice to soften them. In constipation which has existed a long time, in which repeated attacks of fecal colic have recurred, one must bear in mind the possible existence of ulceration and friability of the intestine before manual interference is undertaken.

Psychic, or Suggestive, Treatment.—The treatment of constipation by hypnosis and suggestion has been advocated by Forel, but this method has found few successful imitators.

CHAPTER XVI.

INTESTINAL COLIC—ENTERODYNIA.

In the chapter on the Neuroses of the Intestine a painful affection of the human intestine has been described under the name of neuralgia of the mesenteric plexus, and another under the name of enteralgia, although the two names are practically identical. Under that heading the intestinal neuralgia which occurs in lead-poisoning and which constitutes a genuine neuralgia of the mesenteric plexus was referred to.

By intestinal colic is understood an intense pain which is coincident to, and simultaneous with, a strong tetanic contraction of the bowel. This tetanic contraction may reach such a degree as completely to obliterate the lumen of the intestine. The pain in this condition is caused by excitation of the sensory nerves of the intestinal canal, in consequence of strong contractions of the muscularis, which is not present in enteralgia or neuralgia of the mesenteric plexus. Nothnagel (*l. c.*, p. 72) distinguishes three kinds of intestinal pain: (1) That due to inflammations of the intestinal wall or its peritoneal covering; (2) the genuine nervous enteralgia; (3) the typical colic. Traube, who defined the word colic as meaning the pain which occurs when hollow muscular organs undergo violent peristaltic movements, has almost the same conception as Nothnagel, with this difference, however, that the latter does not simply attribute colic to violent peristalsis, but makes actual tetanic contractions one of the conditions to the pain.

Etiology.

The causes which produce intestinal colic are almost the same as those enumerated in the etiology of enteralgia, the intestinal dystrypsias, and diarrhea, except that they are presumed to exert their effect in a more abrupt and intense manner. Thus, the principal causes are found in errors of diet—substances which are coarse and indigestible or of too cold a temperature (iced water, cold lemonade, mixed drinks, beer, etc.).

The colic which results from overeating was formerly known by the name of *colica saburralis* (*saburra*, meaning sordes, or foul stomach). The colic which is developed in mucous colitis and membranous enteritis has been described in a separate chapter. Frequently it is due to obturating fecal matter, and was then formerly known as "colica stercoralis," although anything that will obturate the intestinal lumen may cause the same characteristic pain—for instance, gall-stones, foreign bodies, and intestinal parasites. The colic which occurs from the development of gases in the intestines, in which the distention may be followed by tetanic contraction, has been described in the chapter on Meteorism. Certain drugs may produce it—for instance, aloes and senna. The colic which occurs in the course of the various anatomical diseases and in the various types of occlusions and enterostenoses has been described in separate chapters. When this painful condition occurs three or four times in one year in the same individual, especially if habitual constipation can be excluded, it should arouse the suspicion of a developing enterostenosis due to pseudoligaments or contracting cicatrices. The suspicion will gain support by the limitation of the pressure pain to one single spot. When the abdomen is exposed to cold externally, it may set up intestinal colic—colica rheumatica.

Symptomatology.

The onset is, as a rule, very sudden, the pain striking the patient in the lower abdominal region, and remaining localized or radiating to other regions. The symptoms vary according to whether it is a transient disturbance, dependent, for instance, upon abnormal formation of gas or obturation by gall-stones, which can usually be relieved promptly, or whether it is associated with a definite anatomical disease, when it will be of longer duration. The form of colic due to the imbibition of cold drinks can also be relieved very promptly. The pains are severe; the patient bends forward or turns over on his abdomen, sometimes pressing both hands into the umbilical region. There is an indescribably anxious and suffering expression on the face; cold sweat gathers on the brow; drawing pains may extend to the rectum and bladder; in severe cases the testicles may be drawn up by the cremaster muscles; the extremities may become cool; the pulse is small and feeble; collapse and fainting may supervene.

Diagnosis.

The condition must be differentiated from biliary, renal, uterine, and gastric colic, and from the intestinal diseases with a definite anatomical substratum. Usually there is a distinct history of the direct cause of the trouble, and the diagnosis is not difficult, although it is not so easy to decide whether we are confronted with a neuralgia of the mesenteric plexus pure and simple, or with an intestinal colic associated with tetanic spasm of the muscularis. For purposes of treatment this really makes very little difference. The types of colic due to an anatomicopathologic substratum require a careful diagnosis.

Treatment.

The treatment consists of removal of the cause, whatever that may be; for instance, the relief of a fecal obstruction or the hastening of the expulsion of a gall-stone or fermenting excess of food. The treatment of the colic due to intestinal parasites is described in a separate chapter; that owing to the prolonged effects of cold on the abdomen requires hot external cataplasms and hot teas, camomile, peppermint, or any good Chinese or Formosa tea internally. The only medicine that will be required is opium—¼ of a grain of the denarcotized extract, one teaspoonful of the camphorated tincture of opium, or from ten to twenty drops of the ordinary deodorized tincture may suffice to cut the attack short if it is not a severe one. Exceptionally, intense colic demands morphin hypodermically. Prolonged and intense attacks may require repeated doses of these medicines. During the attack it is best to abstain from all food and remain in bed.

The acute and chronic nervous enteralgias have been described in a separate chapter.

CHAPTER XVII.

METEORISM—TYMPANITES.

Meteorism means distention of the intestine with gases. It is derived from the Greek word μετεωρίζειν, to elevate. Tympanites is derived from the Greek word τύμπανον, meaning drum, and designates the distention of the abdomen caused by paralysis of the muscular coats of the intestines and their inflation with gas. Curiously enough, both terms do not refer specifically to the intestine, but to distention of the abdomen. Such meteorism occurs in intestinal perforation, where the gas accumulates between the intestine and the abdominal wall. It would be preferable to continue the former exactness of application of the two terms, and apply the word "meteorism" to the acute forms, and the word "tympanites" to the chronic forms, of abnormal intestinal gaseous distention. The two conditions are so frequently described in the clinical part of the work, especially in the introduction to the enterostenoses and occlusions, as well as in the chapters on Dysentery, Carcinoma, Ulcer, Dystrypsia, etc., that in this place the remarks concerning them may be limited.

The intestines contain a certain amount of gas under perfectly normal conditions, and meteorism does not develop although there is a continuous introduction and formation of gas, because the surplus is removed: (1) By eructation; (2) by absorption and excretion of gases through the lungs; (3) by the passage of flatus. In many healthy individuals little or no eructation and evacuation of flatus take place, which is an evidence of the fact that by far the greater amount of intestinal gas is gotten rid of by means of absorption through the intestinal wall and diffusion through the pulmonary gaseous interchange. Tacke (Inaug. Diss., Berlin, 1884) has calculated that in the rabbit from ten to twenty times more of intestinal gases are excreted through the lungs than pass out by flatus through the anus. These experiments are, of course, not directly applicable to the human being, in whom, according to Zuntz ("Verhandl. d. Verein f. innere Med.," Berlin, 84), the anal excretion is at least equally as important as the respiratory. This is the case so far as clinical observations go. Regnault and Reiset were among the first to demonstrate the excretion of marsh-gas by the respiratory air, and to trace its origin to the intestinal canal.

Variety of Intestinal Gases.

A part of the intestinal gas is atmospheric air which is swallowed with the food, and in certain gastric neuroses it is swallowed in the intervals between the taking of food. The oxygen of the atmospheric air is absorbed, and little or none of it is found in the colon. The next most frequent gas is CO_2. Its presence is ascribable to three sources: (1) Diffusion from the intestinal blood-vessels; (2) neutralization of the carbonates in the diet or in the digestive secretions by the HCl of the gastric juice, or under abnormal conditions by the organic acids formed in the course of fermentation; (3) by the fermentation of carbohydrates. The other gases which originate in the course of putrefaction are H, NH_3, CH_4, and H_2S.

As meteorism frequently goes hand in hand with circulatory disturbances, Zuntz was of the opinion that the disproportion between gas development and gas resorption by the blood played the most important rôle in the etiology of this condition ("Deutsch. med. Zeitschr.," 27 u. 30, October, 1884). The opinion had been expressed by Leydig that the intestinal canal participated in the function of respiration. He believed to have found evidences of this function in the intestinal canal of *Cobitis fossilis*, a fish sometimes known as the spine loach, or groundling. Special investigations which were undertaken with regard to this point by J. Paneth could not confirm the assumption of Leydig.*

The chapter on this interesting phenomena is, however, by no means closed, for there really are lower animals that breathe through the intestinal canal (see illustration of tracheal system at the sides of alimentary canal of the *Agrion larva*, Claus' "Text-book on Zoology," English translation by Adam Sedgwick, p. 71). In *Æschna libella* there is also a form of rectal respiration, and the power of the intestinal epithelium in the human being of absorbing gases and excreting them by way of the blood channels through the lungs may be looked upon as a rudimentary function, the last trace of the respiration through the alimentary canal in lower animals.

---

* "Ueber das Epithel des Mitteldarmes von *Cobitis fossilis*," "Centralbl. f. Phys.," Bd. II, 1888, S. 485; No. 24, S. 631. Compare also the results of N. P. Schierbeck, "Sur l'acide carbonique de l'Estomac," "Bulletin de l'Acad. R. des sciences et des lettres du Danemark," 1891; the same author, "Nouvelles recherches sur l'apparition de l'acide carbonique dans l'Estomac," *ibid.*, pour l'année, 1892, Copenhagen, 1893.

Etiology of Meteorism.

Meteorism may be brought about by three conditions: (1) Abnormal introduction of gas into the gastro-intestinal tract; (2) abnormal formation of gas within the intestinal tract; (3) obstructed or diminished evacuation of gas.

1. Abnormal Introduction of Gas into the Gastro-intestinal Tract.—The gaseous distention by abnormal introduction of air is a pathological condition occurring in certain neurasthenic states under the name of pneumatosis or aerophagy. This swallowing of air is sometimes a very incorrigible morbid habit (see author's "Diseases of the Stomach," second edition, pp. 754, 755). It is said that accumulations of gas in the intestine amounting to meteorism may occur from the imbibition of large quantities of liquids charged with CO_2. Personally, the author has never observed meteorism originating from this cause, though it may undoubtedly aggravate any already developing tendency toward this condition, and carbonated waters should not be taken in diseases that are liable to be complicated by meteorism—for instance, enteric fever, appendicitis, peritonitis.

2. Abnormal Formation of Gas Within the Intestinal Tract (Intestinal Fermentation).—This depends upon several factors: (*a*) The amount of fermentable food in the intestine at any time—for instance, the amount of carbohydrate and fats; (*b*) the number and character of the bacteria which are in the intestine; (*c*) the rate of peristalsis. The carbohydrates and cellulose in the diet produce most of the NH_4, whereas the lactic acid, butyric acid, acetic acid, and alcohol fermentation form the hydrogen and CO_2. In all these conditions the diet is a more important factor than the bacteria. Patients subject to meteorism should avoid all vegetables rich in cellulose—for instance, peas, beans, lentils, carrots, turnips, cabbage; all food in which there is liable to be yeast still capable of producing its characteristic biologic action. Sauer-kraut is one of the most disastrous articles of diet for producing meteorism. Some patients exhibit curious idiosyncrasies, and evolve enormous quantities of gas from foods which will be digested with facility in other individuals; thus the author has observed patients in whom stewed plums, and others in whom milk, would produce extreme meteorism. The relation of intestinal fermentation to the bacteria is dwelt upon in the chapter on this subject by Dr. W. R. Stokes.

3. **Diminished or obstructed evacuation of gas** may be brought about by anything that interferes with: (*a*) Eructation of gases; (*b*) discharge of flatus; (*c*) absorption of gas through the mucosa. Interference with eructation may occur in cardiospasm and obstructions of the esophagus and cardia. Diminished or obstructed discharge of flatus occurs in the enterostenoses, the various forms of obstruction, intestinal neoplasms and strictures, any condition leading to paralysis of the muscularis, such as atrophy of the intestinal muscularis, diffuse serous transudation of the muscular layer, such as occurs in peritonitis, etc. The meteorism which is observed in certain infectious diseases, especially in enteric fever, is due to a loss of muscular tonicity. The mechanism of gaseous distention and the so-called obstruction by gas itself—*gasperre*—have been described in the introduction to the chapter on Enterostenoses and Occlusions. The occurrence of extensive meteorism in infectious diseases is generally interpreted as an unfavorable sign, because it signifies a loss of muscular tonus which occurs only in extreme collapse. The immense distention to which the bowel is subject as a result of meteorism in the occlusions constitutes in itself a cause of further gaseous accumulation, because—(1) it inhibits the absorption of gas through the intestinal blood-vessels, and (2) because the excessive distention produces a paralysis of the musculature. The gravest and most extreme forms of meteorism are observed in puerperal peritonitis, for here the paralysis of the intestinal muscularis and the distention of the abdominal musculature permit of the most extensive expansion by the gaseous accumulation. The absorption of gas from the intestinal mucosa is impeded in diseases involving the superficial columnar epithelium.

Meteorism Occurring in Hysterical and Hypochondriacal Individuals.—Psychic affections may produce extensive meteorism in hysterical patients within a very short time. The condition may continue for several days or even weeks, to disappear spontaneously or under the influence of other psychic factors or therapeutic applications, such as abdominal massage or electric treatment. In a number of hysterical patients the author has observed tympanites extending during the entire menstrual period; it has also been observed after parturition. In others it apparently develops and disappears without any assignable cause. The rapid development of such meteorism may be due to neurasthenic paralysis of the intestinal musculature, which may be promptly recovered from, like so many other forms of hysterical paralyses. The rapid evacuation of the gas has been explained by prompt discharge of flatus by way of the anus, which, as Jolly (Ziemssen's "Handbuch d. spec. Pathol. u. Therapie," Bd. XII) suggests, would escape notice because this flatus is composed of odorless gases and could also be controlled in escaping so as not to cause any sound.

Symptomatology and Diagnosis.

Meteorism may be acute, chronic, or paroxysmal, and local or general. One of the first steps in the diagnosis is to differentiate it from ascites, which should not present any difficulties. In ascites the form of the abdomen is distended most in the lateral region when the patient is in the dorsal position, and in meteorism or tympanites the anterior portions of the abdomen are most distended. In patients in whom the abdominal wall is rigid the diaphragm will be forced up very high, causing compression of the lower portions of the lungs and displacement of the heart dullness; and in cases in which the meteorism has developed rapidly, asphyxia, collapse, and even death may result. Palpation reveals an increased tension which may be elastic, like a distended rubber air-cushion, but sometimes is very resistant and hard. Percussion gives a deep, clear, and loud resonance. When conjoint percussion and auscultation is practised, a metallic resonance is elicited which may be confined to single portions of the bowel. Under particularly favorable circumstances this may give a clue to the seat of the obstruction in cases of intestinal occlusion.

During meteorism the intestinal loops possessing the longest mesentery rise to the surface, pressing hardest against the inner side of the abdominal wall. Occasionally the colon is so characteristically distended that it can be recognized by inspection. All conclusions regarding the seat of dilated intestinal loops from inspection, palpation, or percussion should be drawn with great caution, because the most mobile loops are capable of the most surprising displacement by this distention. For instance, the descending colon has been found displaced far over to the right, where we should normally expect the ascending colon, and the transverse colon has frequently been found distended and displaced so as to occupy a position below the umbilicus. Disappearance of the liver dullness, so that on percussion the lung resonance passes over immediately into the tympanitic resonance of the intestines, is frequently observed in these conditions. The excursions of the diaphragm are then so impeded that the respiration becomes exclusively costal, very superficial, and accelerated. In extreme cases the eyes may bulge, and there are dyspnea, cyanosis, and swelling of the jugular veins. Painful sensations are quite constant with this distention, though the characteristic intestinal colic, which, as Nothnagel specifies, always implies a tetanic contraction of the intestine, is rare. In typhoid fever, pneumonia, appendicitis, puerperal peritonitis, meteorism, besides being a very grave sign, may be the direct cause of death. The more rapidly the gaseous distention develops, the more serious are its effects. Quite a number of symptoms usually attributed to the meteorism are really due to the underlying disease and the accompanying nervous effects.

Treatment.

The means of relieving meteorism and tympanites consist of: (1) Efforts to excite the intestinal peristalsis by purgatives or by carminatives; (2) by external abdominal massage, hot or cold friction, counterirritation, or electricity; (3) by mechanical removal of the gas by means of the rectal tube and by large enemata, or, if the case is grave, by intestinal puncture; (4) by efforts to produce absorption of the gas through chemicals administered by the mouth.

1. **Efforts to Excite the Intestinal Peristalsis by Purgatives or by Carminatives.**—In the previous account of the etiology it

has been stated that the bacterial influence is a prominent factor in causing meteorism. This suggests that perhaps an effort at intestinal disinfection would arrest and alleviate the gaseous distention; and, based upon this idea, a perfectly unjustifiable and, to the author's sense, horrible abuse of the so-called intestinal antiseptics has taken hold of some therapeutists. We should not forget that the best intestinal antiseptic that we have is the normal peristalsis of the intestine, and that there is some not as yet clearly defined natural provision for intestinal disinfection which is often injured by the very means we give to destroy the intestinal bacteria. An analogous provision exists in the vagina. Obstetricians have now recognized that disinfection of the vagina by antiseptics during normal labor can certainly work a great deal of harm and rarely effects any benefit, because there are in the female genital passages natural protective provisions against bacterial infection which go a great ways toward preventing the entrance of pathogenic microorganisms. These provisions can be injured by antiseptic irrigations. In a similar manner the human intestine is endowed with a natural means of defense against bacteria which, while we can not as yet exactly define it, we know positively exists (R. Schütz, "Berlin. klin. Wochenschr.," 1900, No. 25). Therefore one of the first principles of treatment is to avoid intestinal antiseptics as much as possible, and first to try the simpler methods of treatment. Among these are the administration of castor oil or aloes. Naturally, when there is an underlying disease which contraindicates the use of purgatives, these can not be employed. The next most available means of treatment, which is particularly effective in the milder cases, are the aromatic carminatives,—camomile, caraway, peppermint, cinnamon, anise seed, fennel seed, cloves,—which are best administered in the form of teas. According to Lauder Brunton, carminatives promote the absorption of gas from the intestinal mucosa. A mixture which has proved of great assistance in the author's experience in the treatment of the so-called flatulent colic is known as the compound mixture of senna:

℞.	Magnesium sulphate,	4	ounces
	Liquid extract of licorice,	1	ounce
	Tincture of senna,	2½	ounces
	Compound tincture of cardamom,	1½	"
	Infusion of senna,	14	"

SIG.—A tablespoonful to a tablespoonful and a half.

A substance has been recommended by Foss ("Deutsch. Arch. f. klin. Med.," Bd. LVI, 126), called "*enterol*," which is said to combine the property of intestinal disinfection with that of a purgative, but the author has not had sufficient experience with this remedy to recommend it. Castor oil and calomel are legitimate remedies in this condition.

We possess no means which can increase the power of the intestinal mucosa to absorb the gases. One can not predict in each case just which purgative will be the most available and effective. Those which have been mentioned, with the exception of the enterol, have been the most helpful in the author's own experience, though in severe cases they have deserted him and he had to have recourse to some of the other methods to be described presently.

2. **External applications, counterirritations, methodical massage, and electricity** all have their proper sphere of usefulness. Naturally, massage can not be used where there is dysentery, typhoid fever, or enterostenosis. When there is no obstruction, electricity in the form of the galvanic and faradic currents combined is sometimes very helpful, especially if one pole is inserted into the rectum by means of a rectal electrode, and the other pole is placed on the abdomen in the form of a broad plate.

3. **Mechanical removal by the rectal tube and large enemata** succeeds when the meteorism is not the terminal symptom of some grave underlying disease that has led to intestinal paralysis and collapse. Enemata of cold water are frequently followed by a striking prompt discharge of gas, very likely by setting up a sudden intestinal peristalsis. Abdominal puncture may become necessary in extreme cases to save life. The technic and criticism of the method are given in the chapter on Enterostenoses. It is important to bear in mind that the puncture must be made by the quickest and most sudden stab movement into the distended loop with the trocar, in order to prevent the escape of the intestinal wall from the pressure of the entering instrument. It should not be used in intestinal paralysis, and if there are symptoms of enterostenoses, the puncture should be followed by a laparotomy.

4. **The efforts to bind the gases chemically** by magnesia usta, prepared chalk, charcoal, or bismuth subnitrate are generally followed by no improvement whatever. This plan of treatment, in the author's opinion, constitutes a waste of time, and sometimes, by deranging gastric digestion, even complicates the clinical situation.

CHAPTER XVIII.

INTESTINAL INDIGESTION—DYSTRYPSIA INTESTINALIS.

Nature and Concept.

By this term is understood any abnormality in the digestive processes occurring in the intestinal canal. The designation "dyspepsia," including as it does the stem of the word "pepsin," should, logically, be used only in reference to such difficulties of digestion in which pepsin is a factor—for instance, in reference only to gastric indigestion. The term "intestinal dyspepsia" is, therefore, not strictly correct. The most important enzyme in intestinal digestion is trypsin, and, accordingly, the author has in a former writing suggested the use of the word "intestinal dystrypsia" for the indigestions which are directly dependent upon disturbed activity of the intestine itself. For those cases which are due to disturbances in the function of the liver or biliary apparatus the designation "hepatic dystrypsia," and for those dependent upon disturbed function of the pancreas the designation "pancreatic dystrypsia," are suggested. These latter terms were first used by Lukjanow ("Allgem. Pathol. der Verdauung"), independently of the author. It is not always an easy matter to locate the symptoms which are interpreted as intestinal dyspepsia exactly in the intestinal canal. What is frequently considered intestinal dyspepsia is, in fact, gastric dyspepsia occurring in a dilated or prolapsed stomach. This makes the importance of chemical and microscopic study of the gastric contents, in such cases, evident. The position and size of the stomach should also be ascertained by one of the methods enumerated in the author's work on "Diseases of the Stomach," second edition, pages 98–113. Intestinal indigestion may be due: (1) To abnormalities of secretion; (2) to abnormalities of absorption; (3) to abnormalities of peristalsis. While the clinical picture of this derangement may be due to disturbance of one of these functions only, —for instance, there are cases where the only demonstrable abnormality is the exclusion of the bile from the intestinal canal (icterus), —in the majority of cases two or all three of these functions are pathological.

The first question to decide is whether the intestinal indigestion is due to an organic, anatomically demonstrable disease, or whether it is purely functional; for the latter may, for instance, occur in the abnormalities of peristalsis, and produce marked suffering from exaggerated or inhibited muscular contractions of the bowel in the total absence of any anatomical lesions.

It is not expedient to classify intestinal indigestion as due to actual anatomical changes, on the one hand, or to functional (neurotic) influences, on the other; nor can it practically be considered under the three types of intestinal dyspepsia due to the disturbances of secretion, absorption, or peristalsis. It is, in fact, still a matter of debate whether a definite classification of the various types of this intestinal disturbance is at all possible or practical in the present state of our knowledge of the intestinal physiology and pathology, for much is still to be learned concerning the interaction of the succus entericus, pancreatic juice, and the bile, and also concerning the changes which occur when the complex gastric chyme mingles with the mixed duodenal secretion. Conscious of the defects of schematization, and not wishing to attribute too much importance to the following classification, it is, nevertheless, offered as the only arrangement that suggests itself to me at present for the purpose of succinctly detailing the various forms of intestinal indigestion. This attempt is all the more justifiable because of a confusing variance of opinion concerning the factors that constitute intestinal indigestion, and the pathological physiology effecting it, as expressed in the American literature of the last ten years. Accordingly, I would suggest arranging the various types of this abnormality in the following order:

I. Intestinal indigestion due to pathologico-anatomical alterations in the structure of the intestinal walls, nerves, lymphatics, or blood-vessels. These are the dystrypsias that occur in association with the various forms of enteritis or enterocolitis, intestinal ulcers, neoplasms, obstructions, stenoses, displacements, etc.

II. Absence or deficiency of the intestinal digestive secretions, especially the bile and pancreatic juice.

III. Intestinal indigestion due to qualitative or quantitative irregularities in the diet. Under this may be distinguished three subtypes: (1) The diet is excessive in quantity and can not be transformed in the normal manner by the amount of digestive secretions present; (2) the diet may be normal in quantity, but irregularly and unhealthfully mixed—there may be too much fat, too much proteid, or too much carbohydrate; (3) the diet contains abnormal injurious substances, either in the form of toxins, the result of putrefaction and fermentation in the diet, or of chemical substances that may have been added by accident or for therapeutic reasons. (Many chemicals and medicines taken for the relief of gastro-intestinal or other diseases exert a deleterious effect upon the normal course of digestion.)

IV. Intestinal indigestion due to abnormal bacterial activity.

V. Intestinal indigestion due to abnormal gastric chemistry.

Thus far the intestinal dystrypsias may be designated as those which arise from abnormalities of the intestinal contents or of the intestinal wall itself. We now come to two forms which are distinct from those hitherto enumerated:

VI. Intestinal indigestion of nervous origin—neurasthenia intestinalis. This should, logically, be classed under the neuroses, but as it is characterized by the one prominent symptom of intestinal dystrypsia, it also demands insertion in this place.

VII. Intestinal indigestion caused by abnormal substances or irritants reaching the intestine from the blood. Under this heading we class the dyspepsias occurring in uremia, malaria, septicemia, croupous pneumonia, erysipelas, influenza, cholera, icterus, diabetes, gout, and uric acid diathesis, the diseases of the blood and blood-making organs, rachitis, osteomalacia, etc.

VIII. Intestinal indigestion due to the activity of intestinal parasites (exclusive of bacteria), worms.

IX. Intestinal dystrypsia due to hyperperistalsis, or excessive motility of the bowel.

Let us now proceed to consider these various forms of intestinal indigestion in a succinct manner, and take them up in the order enumerated.

I. Intestinal Indigestion or Dystrypsia Due to Demonstrable Pathologico-anatomical Alterations in the Structure of the Intestinal Walls, Lymphatics, Blood-vessels, and Nerves.

This series of abnormalities of intestinal digestion has been considered in connection with each of the anatomical diseases to be described in the following chapters of this work.

II. Absence or Deficiency of Intestinal Digestive Secretions, Principally of Bile and Pancreatic Juice.

A correct understanding of this class of intestinal dystrypsias necessitates a thorough knowledge of the physiology of the digestive processes of the stomach and intestine, for which the reader is referred to chapters V, VI, and VII, pages 49 to 70, in the author's work on "Diseases of the Stomach," second edition. Also to the chapters on the Chemical Processes in the Intestine, The Relation of Gastric to Duodenal Digestion, and to the chapters in this work on the Disturbances in the Secretory Functions of the Intestine, Pancreas, and Liver, and Their Influence on the Utilization of Food (Chap. II to VI). Intestinal digestion may be disturbed by abnormalities in the three main secretions—the succus entericus, or intestinal juice, the bile, and the pancreatic juice. Little is known of the abnormal variations in the succus entericus. It contains 0.5 per cent. sodium carbonate, which Bunge regards as an important constituent for the neutralization of the gastric chyme, and if this secretion is lessened or arrested altogether, it is conceivable that the emulsion of fats will be disturbed, and perhaps also the absorption of other food materials will be lessened, because of the partial or complete absence of alkali, and failure to bring about the proper reaction of the intestine.

Disturbances in the Secretion of the Bile—Polycholia and Acholia.—The functions of the liver consist in the production of bile, urea, and glycogen; it participates in the metabolism of the fats and the conversion of certain toxins into harmless substances. Probably the liver is also active in the regulation of the blood circulation. No conservative physiologist will undertake to enumerate all of the probable functions of the liver. Much new material has been published in recent years suggesting new functions, but the results are purely hypothetical. This can be said of the discussion concerning the question whether the liver is capable of influencing the coagulability of the blood after intravenous injection of peptone. This entire polemic is still undecided. The production of bile in the liver-cells is under the influence of a special secretory mechanism depending upon the composition of the food, as well as upon the conditions of the blood circulation in the liver. Direct secretory influences on part of secretory nerves have as yet not been definitely experimentally established. It is probable that nervous influences are active and recognizable only as far as they control the condition of the lumen of the blood-vessels and the rate of the blood flow. A. B. MaCallum, P. Korolkow, and A. von Kōlliker have described nerve branches extending to the liver-cells and even to their nuclei. Contraction of the abdominal vessels from any cause reduces the secretion of the bile; the same results from any causes leading to stagnation of blood in the liver.

Anything that leads to paralysis of the hepatic vessels, causing hyperemia of the liver, at first causes an increase of secretion of bile (Afanassiew and L. Landois). It would lead too far to repeat here all the certain and probable hepatic functions and the influence of bile on intestinal digestion. Even at the present day the part which the bile takes in the work of digestion constitutes the object of active discussion. According to one group of investigators, the bile is simply an excretion, consisting of refuse matter which does not influence the digestive functions. According to others, bile plays a very important rôle in the digestive process. The truth is probably midway between these two extreme aspects. It has been established beyond a doubt that exclusion of bile from the intestinal canal causes such a reduction in the quantity of fats absorbed that only one-seventh to one-half of the amount of fat which is utilized when the bile can enter the intestine comes to absorption (Bidder-Schmidt, Voit, Rōhmann, F. R. Müller, J. Munk). In icterus a considerable reduction of fat resorption has been demonstrated with certainty when there was complete exclusion of bile. According to Rōhmann and J. Munk, the relation between fatty acids and neutral fats is changed in the absence of bile, so that the fat expelled in the feces then consists of from 80 to 90 per cent. of fatty acids (one part of neutral fat to nine parts of fatty acids), while under normal conditions the feces contain only from two to two and a half parts of free fatty acids to one part of neutral fat. The cause of this increase of fatty acids is not known, unless it is due to an excessive bacterial activity in breaking down the fats, the bacteria having an easier task to accomplish this in the absence of the antiseptic action of the bile. The pancreatic juice is capable of splitting up three times as much fat when bile is present as when it is absent (M. W. Nencki). According to R. Fleischer, this is not due to any special fat-splitting ferment in the bile. There is no doubt concerning the influence of bile in increasing the peristalsis, nor concerning its antiseptic action. The hepatic secretion possesses no proteolytic activity. W. Ellenberger and Hofmeister (" Vergleichende Physiologie," 1. Theil, S. 778) state that a mixture of bile and pancreatic juice has a marked proteolytic activity, and Rachford and Southgate (" Medical Record," 1895, p. 878) assert that a mixture of pancreatic juice plus bile plus HCl can accomplish more work in proteolysis than can any other known pancreatic mixture, a statement which has been denied by R. H. Chittenden ("Amer. Jour. of Physiol.," vol. 1, p. 334, "On the Influence of Bile and Bile Salts on the Pancreatic Proteolysis"); B. K. Rachford ("Amer. Jour. of Physiol.," p. 483) concludes that a small quantity of free HCl has little or no retarding influence on the diastatic action of pancreatic juice, but that large quantities of free HCl very materially retard the diastatic action of pancreatic juice. Acid proteids in small quantities slightly increase the diastatic action. Sodium bicarbonate has a very destructive influence on the diastatic action of pancreatic juice. Bile slightly expedites this action. He also reasserts in this paper that bile not only checks the retarding influence which free HCl in large quantities exerts upon the diastatic action of pancreatic juice, but in the presence of free HCl bile very materially expedites the action of the juice. The author has repeated the experiments of Rachford, according to the conditions stated in his paper, and is able to confirm them and agree with him in his main conclusions. In the experiments it will be seen that from four to eight minims of bile were sufficient to neutralize the retarding influence of from four to eight cubic centimeters of a 0.1 per cent. solution of HCl without destroying the acid reaction of the mixture. It is impossible to state the rule regarding the exact amount of bile necessary to neutralize the retarding influence of a definite amount of HCl, thereby giving to a definite quantity of pancreatic juice its greatest diastatic power. But the statements of Rachford are substantially correct. In a similar manner, as bile checks the retarding influence of free HCl, it also has a marked power in diminishing the retarding influence which sodium bicarbonate has upon the diastatic action of pancreatic juice. If there is any free acid in the food as it is discharged from the stomach into the duodenum, the bile will neutralize this acid, and thereby assist the acid proteids resulting from gastric digestion to bring about the most favorable conditions for the diastatic action of the pancreatic juice.

Concerning the diastatic ferment in the bile itself, the opinion has been expressed that it is nothing but the ptyalin which has been absorbed from the mucosa of the stomach and duodenum and reexcreted by the liver in the bile. In view of the powerful amylolytic ferments in the pancreatic juice, the diastatic ferment in the bile can not be credited with important digestive work.

It will be seen, from the physiological factors thus far enumerated, that the chemical processes in the duodenum are extremely complicated, and although by no means completely understood, and much is to be learned concerning them, it is readily seen that they may easily be disturbed. The secretion of the bile, pancreatic juice, and gastric juice may vary abnormally, and throw out of balance the finely adjusted chemical transformations which go on in the upper intestine, giving rise to symptoms of dystrypsia, and eventuating in disturbances which oftentimes can not be traced from the purely clinical standpoint, and for the elucidation of which we depend upon future chemical and physiological investigation.

Polycholia—Overproduction of Bile, Together with All its Constituents.—The bile pigments and bile acids owe their origin to the functions of different cells (O. Hammarsten), and can be increased or diminished independently. Poisons that injure red corpuscles cause an increase in the pleochromatic substances (the pigments of the bile)—for instance, ether, chloroform, toluylendiamin, arsenuretted hydrogen. Under their influence the hemoglobin, becoming free from the erythrocytes, is taken up by the hepatic cells and changed into bilirubin. The hemoglobin as it becomes free of the erythrocytes may pass over into the bile without becoming changed into bile pigment. Phosphorus increases the production of bile pigments (E. Stadelmann). According to M. M. Tschelzow, the extract of chionatus virginica is a powerful cholagogue, but the importance and reliability of so-called cholagogues have been much weakened by investigations of Baldi, Paschki, Mayo Robson, and Nissen.

The dietetic cholagogues deserve most confidence, and the effect of a number of foods in this respect is really beyond a doubt. S. Rosenberg, for instance, has demonstrated the fact that olive oil increases the secretion of bile, while the concentration diminishes.

Acholia.—Diminution of the production of bile in its entirety or in its single constituents has been observed: (1) While in a state of starvation or hunger; (2) during febrile diseases; (3) uremia (Lukjanow, *l. c.*, p. 151), the bile becoming in the latter case poorer in water; (4) reduction of the size of the liver by resection. Liver of guinea-pigs reacts to ligation of the common gall-duct by necrosis of hepatic cells—*i. e.*, a reduction of the liver parenchyma (an analogous condition results in atrophic liver cirrhosis). Chemical agents—potassium iodid, calomel, iron, copper, atropin, toxic doses of strychnin, and alcohol—reduce the production of bile.

Less bile reaches the intestine when there is obstruction by gall-stones, cholelithiasis, catarrhal icterus, neoplasms compressing the common gall-duct, or floating kidney.

A. von Haller ("Elementa Physiologiæ," 1, p. 615) calls attention to bile entering the intestine in its upper part in all animals, which, he claims, is a sign of its special importance for digestion, and it can not, therefore, be an excretion simply; if it were, it would surely have been conducted outward by an outlet nearer the rectum.

The common gall-duct meets the pancreatic duct within the walls of the duodenum. Either a common duct is formed by the union of both, or the two ducts open into a special receptaculum—the diverticulum of Vater; a wart-like prominence in the mucous membrane of the duodenum marks the place where this diverticulum opens. Irritation of this spot constitutes the signal for the outflow of the stored bile and pancreatic juice. This anatomical arrangement suggests that the combined action of the pancreatic juice and bile is contained in the plan of the digestive process. The doctrine of the detrimental effects of polycholia or acholia is not based upon succinct and incontrovertible proofs. There are many defects in the experimental and clinical logic, because affections of the liver are, under natural conditions, always complicated by diseases in other parts of the body, and clinical investigations have not succeeded in separating the results due to disturbed bile formation from the totality of symptoms in an unobjectionable manner. In chapters II and VI has been emphasized the interesting phenomenon that, in the gastro-intestinal tract, one digestive function may vicariously be replaced by increased function in another part of the apparatus; for instance, increased hyperacidity of the gastric juice may be counterbalanced, to a certain extent at least, by increased alkalinity of the pancreatic juice or succus entericus.

When we consider the effects of absence of bile on digestion, we must also bear in mind the systemic effects which bile intoxication may bring about under those conditions. At the present day most pathologists assert that all forms of icterus are of hepatogenic origin; and in those diseases in which there is no stagnation of bile, the icterus is explained by presuming that the hepatic cells have lost their power of holding back the bile, which consequently diffuses into the juices of the body. This form of icterus has been designated "diffusion icterus," in contradistinction to that due to obstruction of the bile-ducts, which is known as "stagnation icterus." In diffusion icterus we are not confronted with a poisoning of the body by bile constituents, but rather with a poisoning with those toxic substances which should have been rendered innocuous and been reconstructed in the liver—*i. e.*, in diffusion icterus not the secretory functions, but the chemical and metabolic functions, are injured. These facts are merely emphasized here in order to make plain the complex question with which we are dealing, and to show how difficult it is to separate the purely digestive disturbances from the systemic and metabolic disturbances.

Antizymotic Influence of the Bile.—The antiseptic effect of the bile, which was first prominently emphasized by Frerichs in 1846, has since received much support from Fleischer, Maly, and Emich. The degree of intestinal putrefaction can be determined by the increase of the total ethereal sulphates in the urine (E. Baumann). In a number of cases of purely catarrhal icterus the result of gastroduodenitis, E. Biernacky has demonstrated that the amount of the ethereal sulphates and their ratio to the preformed sulphuric acid increase. The important question for the clinician in this connection is whether he can obtain any tangible indication from urinalysis which will instruct him regarding the degree of putrefaction in the intestine. On the basis of a very large experience the author is able to express the opinion that the amount of total sulphates and the amount of indican in the urine, irrespective of the ratio of the ethereal sulphates to the preformed sulphuric acid, constitute such an available diagnostic indication. Other means shall later on be suggested for obtaining information regarding abnormalities in the chemical functions of the upper intestine.

Disturbances in the Secretion of Pancreatic Juice—Succorrhœa Pancreatica; Hypochylia and Achylia Pancreatica.—The secretion of the pancreatic juice is dependent upon the nervous system; the secretory fibers for the pancreas are, according to J. P. Pawlow, contained in the vagus. The pancreatic secretion contains four enzymes: trypsin, amylopsin, steapsin, and a milk-curdling ferment (Halliburton and Brodie). R. Lépine assumes the existence in it of a sugar-decomposing ferment.

Succorrhœa Pancreatica.—We may assume a pathological increase of the pancreatic juice when the secretory innervation of the gland is exaggerated. Bernstein has recognized, after cutting the nerve that accompanies the larger arterial branches, that the secretion continues and is even increased. There is also a so-called paralytic pancreatic secretion. There is no doubt in the author's mind that neuroses of the pancreas exist in which the secretion of the gland is pathologically increased or decreased, or may be altered in its chemical composition without being either augmented or diminished. We know that the salivary glands can be influenced by diseases of remote organs, thus producing a reflex salivation. It is probable that a reflex succorrhœa pancreatica exists, for irritation of the central end of the lingual nerve and irritation of the central end of the vagus increase the pancreatic secretion.

Duodenal Test-meals.—In the second edition of "Diseases of the Stomach," page 55, the author has narrated the case of a female patient who had suffered from repeated attacks of biliary colic. She had passed small stones without giving her much pain. By duodenal intubation he aspirated a clear but viscid and sticky fluid similar to egg-albumen, containing no bile pigments nor cholesterin. It was capable of digesting fibrin and serum albumin in an alkaline solution. It also possessed amylolytic and fat-decomposing properties. He concluded that it was almost pure pancreatic juice, and that the gall-duct was stenosed by a small calculus sufficient to prevent the influx of the bile into the duodenum. This experience suggested the possibility of duodenal test-meals—*i. e.*, of aspirating test-meals from the duodenum after weighed amounts of proteids, carbohydrates, and fats had been given, and determining the adipolytic, amylolytic, and proteolytic power of the filtrate. The author has analyzed duodenal test-meals with this object in view. Before attempting the aspiration of test-meals from the duodenum (Hemmeter, "Versuche über Intubation des Duodenum," "Arch. f. Verdauungs-Krankheiten," Bd. II, S. 85), it is necessary to obtain a knowledge of the state of the gastric chemistry. The amount of free and combined HCl and the total acidity, the amount of acid salts and organic acids, the state of gastric amylolysis, should be determined beforehand, for it will make a great and important difference in the total acidity of the duodenum whether an individual has hyperchlorhydria or achylia gastrica in his stomach. The next most important preliminary knowledge is to know just when to intubate the duodenum, preferably by Kuhn's method—*i. e.*, how long after the completion of the gastric digestion. This can be ascertained only by several tests of the rate of gastric peristalsis in the same patient. For instance, if we have found out that the stomach is capable of expelling a meal consisting of four ounces of Hamburg steak, two ounces of boiled rice, and three ounces of bread and butter in four hours, we should make our preparations for duodenal intubation about three and a half hours after the ingestion of this meal. Then comes the indispensable determination of the emptiness of the stomach, which is done by allowing eight ounces of warm water to run in and siphoning it out again. By this procedure we can very soon discover whether the stomach is empty—if it is empty or nearly so, we may proceed to make the attempt to intubate the duodenum. The plan seems to be confronted with insurmountable difficulties at present, but it is a method to which we are surely coming, for in the author's opinion it constitutes the only reliable means for obtaining any information regarding the chemical processes of the duodenum (see chapter on Duodenal Intubation).

Certain chemical substances may cause increase in pancreatic secretion—for instance, pilocarpin. There are certain diseases of the pancreas which produce an augmented secretion (Lukjanow, *l. c.*, p. 198).

Hypochylia and Achylia Pancreatica.—Diminution or absence of the pancreatic secretion can be caused by a variety of conditions: (1) Partial or general atrophy of the gland tissue; (2) diminished secretion due to nervous influences; (3) diminished secretion in anemic conditions, subnutrition, and general weakening of the organism; (4) diminution of pancreatic secretion due to chemical substances (atropin, toxic doses of strychnin); (5) diminished secretion due to febrile processes. The experimental diminution of pancreatic secretion produced by the artificial production of fever is so intensely interesting that it merits a brief consideration. The experiments were conducted by J. J. Stolnikow (quoted by Lukjanow, *l. c.*, p. 200). He found, after producing fever artificially in dogs by septic infection, that the amount of pancreatic juice had increased from 30 c.c. to from 70 to 79 c.c. an hour; it is followed by a marked diminution of the secretion, which is of long duration and very obstinate. When the fever had lasted for from two to ten hours, the extracts made from the secretion of the gland were much more energetic than the normal ferments. But if the fever lasted a long time, they became weaker in action. Stolnikow attributes the variations in the amount of ferments to changes both in the pancreatic cells, as well as in the trophic nerve apparatus. But the most interesting deduction, however, from these experiments was that which gave evidence of the inequalities in the variation of the amounts of proteolytic, diastatic, and adipolytic ferments secreted—while the adipolytic and diastatic were much reduced during the fever, the amount of the proteolytic ferments remained at a considerable height. In addition to this Stolnikow could establish that the amount of the three different ferments was influenced by the kind of food which predominated in the diet. This experimental material justified Stolnikow in the conclusion that there must be three separate nerve mechanisms which control the production of the three ferments of the pancreas. These experiments permit of an insight into the intricate neurosecretory mechanism of the pancreas.

Alkalinity of the Pancreatic Juice.—According to J. P. Pawlow (Wratsch, 1893, S. 1232, Russian), a dog secretes just enough pancreatic juice in the course of a day as is necessary to neutralize the amount of gastric juice secreted. As this conclusion is more or less applicable to the human physiology, we may readily understand how intoxication with the HCl of the gastric juice may occur in those cases of stenosis of the pancreatic duct or atrophy in which no pancreatic secretion is poured into the duodenum.

Systemic or Metabolic Effects Produced by Experimental or Pathological Suppression of Pancreatic Juice.—In some animals the ligation of the pancreatic duct is tolerated comparatively well. According to Heidenhain, the proteolytic ferment is reconverted into the harmless zymogen. In other animals, however,—for instance, pigeons,—the general nutrition suffers severely. Dogs, rabbits, and cats show no evil results. We may assume, therefore, that when the pancreatic duct is stenosed, the system becomes deluged with pancreatic secretion, which continues to be formed for a while, at least, and a condition may result which can be designated, for want of a better term, as "pancreatic icterus,"* which is harmful in some animals and borne well by others. Whether or not the "*pancreatic icterus*" is deleterious to the human organism we are not as yet able to judge. But there are other dangerous metabolic effects which result from extirpation of the pancreatic gland. These consist in the development of a genuine diabetes mellitus, first experimentally produced by J. von Mering and O. Minkowski. It is plain, therefore, that the pancreas, in addition to the secretion that it pours out in the intestine, has also an internal secretion amounting to the formation of a glycolytic enzyme which effects the destruction of sugar in the blood (Lépine and Barral). This can serve to explain the striking difference in the consequences of complete extirpation of the gland on the one hand, and simple ligation of the duct, on the other. After the extirpation of the gland the glycolytic enzyme must, naturally, be wanting, and therefore the destruction of sugar can not be effected. Later investigations of M. Arthus, and also of Minkowski and J. von Mering, suggest that the glycolytic enzyme does not act by way of the blood.

---

* An objectionable term, because the word icterus refers to a color substance which the pancreatic juice does not contain; we are using the expression only conditionally.

This entire discussion illustrates the difficulty here, as well as in connection with hepatic dystrypsia, of distinguishing between the effects produced by simple absence of the special digestive secretion from the intestinal canal and the systemic or metabolic effects produced by diseases of the bile-ducts and liver on one hand, and the pancreas or pancreatic duct, on the other.

What are the consequences of disturbed secretion of pancreatic juice? It is not probable that excess of pancreatic secretion—succorrhœa pancreatica—could produce any marked disturbances, but diminished pancreatic secretion, hypochylia or achylia pancreatica, reduces the digestion of the fats. F. R. Müller has had opportunity to study several cases of more or less complete degeneration of the pancreas in human patients. While, normally, 84 per cent. of the fats were transformed into glycerin and fatty acids, in these patients only 40 per cent. were transformed. The digestion of proteids was reduced but slightly, and the assimilation of carbohydrates was not diminished at all. It is probable that some of the pancreatic tissue was still preserved in Müller's patients, for in animals in which M. Abelmann removed the pancreas completely, the entire fat that was ingested appeared again in the feces. This seemed to be due to removal of the gland itself, for if pig's pancreas was given in the food of such animals, they absorbed considerable of the fat in the diet. The same can be said when artificial emulsions of fat were given. Natural emulsion, or milk, was absorbed to the amount of 53 per cent. of the fat after extirpation of the pancreas. The proteids are absorbable to the amount of 44 per cent. after complete extirpation, the amount absorbed being increased by the ingestion of pig's pancreas. Von Recklinghausen and Hoppe-Seyler have observed that the chylous vessels were filled with white chyle in cases of almost complete degeneration of the pancreas, and the latter author assumes that the formation of soaps and emulsification of fats are brought about by bacteria under these conditions.

Is there any way in which we can study the alterations in the pancreatic and hepatic secretions? So far as diseases of the pancreas are concerned, they have hitherto been of subordinate clinical interest only; *there is not a single symptom or objective sign which could be referred to a disease of the pancreas with certainty.* (See Oser, "Erkrank. d. Pankreas," in Nothnagel's "Spec. Pathol. u. Ther.," Bd. XVIII; also A. W. Mayo Robson, "Pancreatitis," "Phila. Med. Jour.," June 1, 1901, p. 1048.) It is, therefore, very difficult to diagnose these diseases during life. They are also frequently complicated with other diseases. This is true especially of stenoses of the duct. Both ducts,—*i. e.*, the pancreatic duct and the hepatic duct,—for anatomical reasons, are frequently occluded simultaneously. It is, therefore, impossible to assign special symptoms to the pancreas or liver, from a clinical standpoint; in addition, the pancreatic functions may be vicariously supplanted and compensated by altered activity of the remaining glandular apparatus.

There are, in the author's opinion, but two ways in which any progress can be hoped for—one is by duodenal test-meal analysis, described in the preceding, and the other is by careful quantitative analysis of the stools after weighed amounts of proteids, carbohydrates, and fats have been ingested, not only for the purpose of determining the residual undigested amount of each of these substances, but also for determining the amount of proteolytic, amylolytic, and eventually also of adipolytic ferments present in the feces by the same method which the author utilized in determining these ferments in the human feces (Hemmeter, "Ueber das Vorkommen von proteolytischen und amylolytischen Fermenten im Inhalt des menschlichen Kolons," "Pflüger's Arch. f. d. ges. Physiol.," Bd. LXXXI, S. 151). From the amount of undigested food of the various classes and the quantitative determination of the various ferments, a fairly reliable conception of the state of pancreatic secretion should be obtained. It must be emphasized, however, that the determination of the ferments must be made under antiseptic precautions such as were applied in the article quoted (*l. c.*). The Boas stool-sieve is a rough but quick and practical method for arriving at an approximate conclusion concerning the amount of undigested food (see chapter on Examination of Feces). This entire subject is of immense importance, and demands systematic and accurate methods of diagnosis for the recognition of a group of diseases—the hepatic and pancreatic dystrypsias—on which our knowledge is still in its infancy.

Hemorrhagic Pancreatitis Caused by Obstruction of the Outlet of Vater's Diverticulum by Gall-stone.—Experimental pancreatitis can be produced by many substances when injected into the pancreatic duct, and it seems as though the element of infection plays an insignificant, if not an entirely unessential, part in the production (S. Flexner, "Contributions to the Science of Medicine," dedicated to William H. Welch by his pupils, 1900). Simple ligation of the pancreatic duct and absolute retention of pancreatic secretion within the gland and its excretory ducts produce different results in different animals (Pawlow and Lukjanow). In some animals—the rabbit, for instance—a diffuse proliferation of the connective tissue and degeneration of the glandular parenchyma take place. The dilation in the excretory ducts caused by the obstructing ligature may give rise to cystadenoma (Treuberg).

But only recently has the experimental evidence been furnished that bile, when injected through the pancreatic duct into the pancreas, produces at once a very extensive acute hemorrhagic pancreatitis. Dr. Eugene L. Opie has proved that bile can produce such pancreatic lesions in the absence of infectious agents (American Surgical Association Session at Baltimore, May, 1901; see "American Medicine," May 18, 1901, p. 277). This discovery is of importance because of its bearing upon stenosis of the diverticulum of Vater by gall-stones, for small gall-stones may partially or completely plug up the opening of the diverticulum at the duodenal papilla and thus cause hemorrhagic pancreatitis by forcing the bile to flow into the pancreatic duct. At the same meeting of the American Surgical Association Mr. A. W. Mayo Robson, of Leeds, England, gave the clinical evidence that a gall-stone lodged in the common duct just above the diverticulum of Vater or completely plugging up the diverticulum produces obstruction not only to the biliary, but also to the pancreatic, secretions, which, in the pancreas, leads to chronic interstitial pancreatitis. Clinically this condition is associated with great loss of strength and weight. The pancreas assumes an enlarged, hardened, and indurated shape. In 1899 I made an autopsy at the Maryland General Hospital on a male patient who had died after a slight attack of jaundice, epigastric pain, and bile in the urine, but who had rapidly emaciated. A hard, nodular tumor was palpable at the edge of the liver, extending to the pylorus. As there had been absence of HCl in the gastric contents and greatly impaired peristalsis with excess of lactic acid, the diagnosis was made as carcinoma of the pylorus. The autopsy showed a diffuse interstitial pancreatitis; the pancreas was hard, indurated, and had given the sensation of a tumor to the palpating fingers. In the diverticulum of Vater was a gall-stone the size of a small cherry; in the gall-bladder were 15 gall-stones of various sizes, ranging from that of a cherry to the smallest grains. Adhesions existed between the pancreas, liver, and pylorus. The case also showed disseminated fat necrosis and, histologically, many small areas of hemorrhage could be detected in the pancreas. The duct of Wirsung contained bile. A case very similar to this is reported by Opie from Halsted's clinic at the Johns Hopkins Hospital. Such cases give evidence of the wide-spread damage that may arise from gall-stones impacted in or near Vater's diverticulum, and in cases of supposed intestinal indigestion with rapid emaciation and a history of cholelithiasis the practitioner will have to think of these possibilities and urge surgical treatment. From a prognostic standpoint the condition is as grave as that of carcinoma of the pancreas, gall-bladder, or liver, for which it is readily mistaken, but prompt operation and removal of the stone generally lead to recovery. Cholecystotomy and drainage of the gall-bladder relieve the tension and allow of absorption of the exudate, and the chronic interstitial pancreatitis disappears and the duct becomes patent again, the intestinal dystrypsia ceases, and the patient gains weight.

III. Intestinal Indigestion Due to Qualitative or Quantitative Irregularities of the Diet.

The results of the three subtypes under this heading—(1) The diet too excessive for the digestive secretions to transform; (2) a diet which may perhaps be normal in quantity, but unhealthfully mixed, so that it may contain too much fat, carbohydrate, or proteid; (3) diet which already contains food in a state of putrefaction and fermentation—have already been sufficiently considered in the chapters on Diarrhea, Enteritis, and Dysentery; the chapters on Diet and Prophylaxis and Hygiene by Diet also refer to this subject. In this connection we must emphasize the injurious effects of hasty eating, because it prevents proper mastication and insalivation of the food, and brings the latter into the stomach in a state which will later on set up irritation, and also because the digestive juice can not penetrate morsels of large size, and also because a certain amount of amylolysis is expected to take place in the stomach, and can not do so in the absence of proper insalivation.

Dystrypsia Caused by Medicines.—Not sufficient attention is paid to the great harm that is done to the patient by medicine. Many drugs which are given for the very purpose of relieving supposed indigestion and dystrypsia are actually the causes of it. Here may be mentioned all condiments, particularly pepper, mustard, and ginger. Among the drugs that have done great harm in the author's experience are the bromids, salicylate of soda, all astringents, the bismuth salts, iron, when not given immediately after meals, belladonna, bicarbonate of soda, HCl, and pepsin. The latter drugs should be given only after test-meals showing either a hyperacidity which calls for alkalis, or achylia gastrica which demands HCl. Innumerable are the cases which have presented themselves to the author for treatment and who were taking HCl when the stomach was already forming an excess of that acid. Among such patients were quite a number of physicians.

IV. Intestinal Indigestion Due to Abnormal Bacterial Activity.

This subject has already been exhaustively treated by Dr. Wm. Royal Stokes in the chapter on Intestinal Bacteria, and the essential features have been recapitulated under the various diseases in which the bacterial activity is morbidly increased. A. Czerny, P. Moser, Bednar, and Henoch, in Germany, have asserted that a large number of the indigestions and diarrheas of children depend upon fermentative and putrefactive processes in the gastro-intestinal canal, and generally without anatomical changes in the mucosa. The instructive investigations of Wm. D. Booker ("Johns Hopkins Hospital Reports," vol. VI), while recognizing that the gastro-enteritis in children is a general infectious disease, demonstrate also that there are considerable anatomical alterations in the mucosa (see Hemmeter's "Diseases of the Stomach," second edition, pp. 423 and 424). The bacterial flora becomes exuberant and harmful in its activity, as a rule, only after grave errors in diet have been committed. This is true both in infants and adults. The immediate results of this state are abnormal putrefactions and fermentations, with the formation of toxic and irritating substances which have a deleterious influence on the mucosa, and may set up enteritis and colitis. All this has already been considered in the chapters on Diarrhea, Dysentery, and Enteritis.

It was an opinion, first expressed, probably, by Pasteur, that normal intestinal digestion could not be carried on in the total absence of bacteria. In the experiments of Nuttall and Thierfelder young guinea-pigs taken from the pregnant mother by cesarean section were kept under such aseptic control from the first moments of life so that the entire alimentary tract was entirely free from micro-organisms, and the feces were sterile (see chapter on Intestinal Bacteria). It was concluded that the bacteria were not essential to the digestion and life of the animals. (See also the same author's "Animal Life without Bacteria in the Digestive Tract," third communication, "Experiments on Hens," "Zeitschr. f. physiol. Chem.," Bd. XXIII, S. 231, 1897; also N. W. Nencki, "Digestion without Bacteria," "Arbeiten des Vereins Russischer Aerzte," St. Petersburg, January, 1896.*)

Similar experiments have been carried out by Schottelius ("Arch. f. Hyg.," Bd. XXXIV, S. 24), but instead of selecting the guinea-pig, hens and their eggs were utilized for the experimental work. Two sets of eggs were taken, one in which the surface was made absolutely free from bacteria, the other set remaining contaminated with micro-organisms. The first will be called, for convenience, sterilized eggs, while the other batch will be designated as nonsterile eggs. The former were introduced into a sterile incubator, while the others were not. When the chickens were hatched, those from the sterilized incubator were carefully fed on sterilized food, while those of the control experiment were given ordinary food. The feces were examined in both instances. In the so-called sterile chickens the feces were found to be absolutely free from all forms of bacterial life, while the reverse was found to be the case in the control experiment. At the end of a certain period of time the nonsterile batch of chickens was found to have gained in weight much more rapidly than the sterile batch, the gain being 250 per cent. greater in those in which the bacteria gained access to the alimentary tract. All the so-called sterile chickens died within three weeks after they had been hatched, while the control chickens lived on as usual. This suggests that the presence of a certain form of bacteria is essential to the perfect functioning of the digestive process in the gastro-intestinal canal. In connection with these experiments of Schottelius it is of importance to add that the shell of eggs, like the human skin, can not be thoroughly sterilized. There always will remain bacteria in the deeper layers resisting the influence of antiseptics.

---

* For further reference to digestion without bacteria see G. H. F. Nuttall and H. Thierfelder, "Thierisches Leben ohne Bakterien in Verdauungskanal," "Zeitschr. f. physiol. Chem.," Bd. XXI, 1895–96, H. 2 u. 3, S. 100; Bd. XXII, 1896–97, H. 1, S. 62; "Weitere Untersuchungen über bakterienfreie Thiere" (vorgetragen von Hrn. H. Thierfelder). "Archiv von Du Bois-Reymond," 1896, H. 3 u. 4, "Verhandlungen der Berl. physiol. Gesellschaft," S. 363.

The interesting observations of Busch on a woman thirty-two years old who had received a fistula just below the duodenal papilla in an accident have demonstrated the possibility of digestion in the absence of secretions. The entire duodenal and gastric secretions escaped by means of the fistula. She emaciated alarmingly in the course of six weeks, the pulse became very slow, she was weak and somnolent, and food taken by the mouth did not satisfy her appetite. When Busch introduced the food into the lower opening communicating with the fistula, it was digested and she gained in weight. The food was inclosed in gauze sacks which could be withdrawn by threads. The digestion must, in this case, have been effected by bacteria. The case has been more fully described in the chapter on Intestinal Bacteria by Dr. Wm. Royal Stokes. Busch's original case is described in "Virchow's Archiv," Bd. XIV, S. 140.

It has been suggested that the bacterial acetic acid and lactic acid fermentation might be able to increase the total acidity of the intestinal contents to a degree in which certain pathogenic microbes could not exist, and, as a further reason for assuming that bacteria may be useful, their ability to produce peptone and albumoses, in fact, similar products as result from peptic and tryptic digestion, has been emphasized. The whole question of the normal or abnormal conditions that may accompany bacterial activity in the intestines, and what really constitutes normal and abnormal intestinal bacterial activity, is as yet undecided. The experiments of Nuttall and Thierfelder are, of course, not directly applicable to the human being; neither are those of Schottelius. In no case were the experiments conducted sufficiently long to prove conclusively that even the animals on which they were carried out could exist indefinitely with an absolutely sterile gastro-intestinal canal. Concerning the increase in the total acidity, which could be brought about by some bacteria and might be injurious to other pathogenic organisms, it might be wise to remember that what is injurious to pathogenic bacteria within the intestine, and sufficiently toxic to kill them, may also be injurious and toxic to the general organism of the individual, and especially to his intestinal mucosa.

In the treatment of these bacterial processes it is not well to place too much dependence on the so-called intestinal antiseptics, but, above all things, to give the utmost care to the diet—let it be easily digestible, thoroughly sterilized, and moderate in quantity. Sterile milk, egg-albumen, if need be mixed with wine or brandy, and scraped beef, slightly broiled or even raw, as it can be prepared perfectly sterile in that condition, would answer the purpose for the first week. Thereafter broiled Hamburg steak and sterile milk and bread will answer the demands of the organism. When the bacterial action of the intestine is excessive, it is wise to avoid egg in the diet. An extensive experience in the dietary management of this condition has led the author to exclude them. The dangerous products that can be formed from egg are lecithin, and from this cholin, and the highly poisonous neurin. The possibility of the formation of these substances from eggs and lecithin is based on the work of Bokai ("Zeitschr. f. physiol. Chem.," Bd. 1, S. 157). Next to a careful diet the most effective means for combating bacterial activity and autointoxication are lavage of the stomach and colon. Generally nothing is needed but sterile water. If there is no HCl in the secretions of the stomach, a solution of this acid—3 : 1000—is advantageous. For lavage of the colon a semisaturated solution of thymol exercises sufficient antibacterial properties, but even this is rarely necessary.

V. Intestinal Dystrypsia Due to Abnormal Gastric Chemistry.

That abnormalities in the gastric secretions may bring about intestinal dystrypsia is well known from the intestinal meteorism and disturbances of peristalsis—both hyper- and hypoperistalsis—which follow upon gastric hyperchlorhydria, and the frequent diarrheas observed in connection with achylia gastrica. Analysis of gastric contents is indispensable if we wish to understand intestinal dystrypsia in all its phases. If we find hyperchlorhydria, sufficient alkalis must be given to neutralize the excess. A diet should be selected according to the principles laid down in the author's work on "Diseases of the Stomach," second edition, page 828. The amount of alkali to be given can be determined according to the principles laid down in the same work on page 337. In all cases where there is absence of free HCl (anacidity, achylia gastrica) the author has found it useful to give HCl in double gelatin capsules, as recommended by C. O. Aaron, immediately after the meal. Some stomachs which have not secreted HCl for a long time become so sensitive to this acid that the patients are conscious of gastric distress every time it is taken. In those cases the author has begun by giving one drop of the official dilute acid after each meal and gradually increasing the dose.

VI. Intestinal Dystrypsia of Nervous Origin (Neurasthenia Intestinalis).

In the various parts of this work the prominent influence of the nervous system on the secretion of the digestive juices has been emphasized. It is, therefore, not surprising that there should exist a state of intestinal dystrypsia of purely nervous origin which can not logically be classified under any of the other headings. Rosenheim ("Krankheiten des Darms") considers it a combination of various intestinal neuroses depending partly on functional weakness and partly upon exaggerated irritability of the vagosympathetic fibers of the intestine. It generally accompanies neurasthenia gastrica. The patients complain of distention, fullness in the abdomen, pain, sometimes borborygmi, sometimes painful desire for stool, which is not followed by an act of evacuation. There are some sufferers from intestinal neurasthenia who seem to have no gastric symptoms, for they seem quite well immediately after eating and for two or three hours after meals. Then they begin to complain of distention of the abdomen, accelerated heart action, and sensations of heat or cold; sometimes they find it impossible to lie down, but seek relief by moving about restlessly, rubbing their abdomen. The condition of the evacuations is variable. The majority of such patients, in the author's experience, suffer from constipation associated with meteorism. But there are others in whom diarrhea is a symptom, and it generally occurs between midnight and two o'clock in the morning. It is characteristic of these conditions that the distress very frequently ceases spontaneously after several hours, which is an evidence that the dystrypsia is a functional weakness. Both the constipated cases of neurasthenic dystrypsia, as well as those in which night diarrhea is a symptom, exhibit a very capricious behavior of the intestine, for constipation and diarrhea may alternate in the most surprising manner and without any assignable cause. The state of the bowels is independent of the nature of the diet. At times the most indigestible food taken in large quantities is excellently digested. At other times the most careful diet is followed by distress.

One often observes that intestinal neurasthenia occurs in patients who are habitually addicted to overeating, but nevertheless they remain emaciated and weak. This is not dependent on any diarrhea. In a large majority of these types there are evidences of intestinal autointoxication and excessive bacterial activity, or there is a clear history of uric acid diathesis and general neurasthenia.

Diagnosis of Neurasthenia Intestinalis. — This depends, above all, upon the exclusion of demonstrable intestinal diseases associated with anatomical lesions—the symptoms of abdominal distress, independent of the diet; the presence of constipation or night diarrhea; or of the most bizarre alternations of diarrhea and constipation in the absence of enteritis or colitis; the anamnesis of general neurasthenia, hysteria, hypochondriasis, uric acid diathesis, and autointoxication.

Treatment.—(1) The diet. In the author's experience it is impossible to succeed with a radical enforcing of strict diet of any kind, but it is wise to recommend what is known as a bland diet; above all things, to study the stools, if possible by the stool-sieve, in order to discover what foods pass the intestinal tract undigested. These must, thereupon, be avoided, or given in so finely divided a state and in such small quantities that their digestion becomes easier. It is also of importance to utilize the observations which the patients have made upon themselves with regard to the diet. They frequently know what causes distress and what can be digested. The stool examination, however, will guard against deception. The author makes it a rule to forbid alcohol, tea, coffee, and tobacco to these patients. In great exhaustion he allows small quantities of a superior wine or whisky, under strictest control. In some patients a diet rich in carbohydrates, in others one rich in proteids (beef, fowl, fish), is well digested. In fact the diet to be followed can not be decided until a dietetic experiment is made. (2) The same holds good with regard to the treatment of the constipation. Here the practitioner will have to decide whether this is due to atony or spasm of the intestinal musculature, for what will benefit one condition will harm the other. Here, also, in most cases, a therapeutic experiment will be necessary before we can discover whether we are confronted with atony or spasm. The majority of these patients require exercise after meals. A few that are weak may require rest in bed. Especial attention must be paid in the selection of the food for the last meal of the day. Great moderation in the amount eaten and digestibility are the most important points to bear in mind. A heavy supper will often produce a restless night. (3) The treatment of the general neurasthenia will be that by electricity, hydrotherapy, massage, and baths, as detailed in the chapter on Gastric Neurasthenia, "Diseases of the Stomach," second edition, page 872.

VII. Intestinal Indigestion Caused by Abnormal Substances or Irritants Reaching the Intestine from the Blood.

That toxins may be excreted from the blood through the walls of the intestine has already been referred to in the chapter on Intestinal Ulcers, for this factor constitutes one of the elements in the etiology of intestinal ulcers. Their occurrence has there been described as a result of uremia, septicemia, gout, scurvy, leukemia, and in consequence of certain poisons excreted through the intestinal mucosa—mercury, for instance. The intestinal ulcers which have been observed in consequence of extensive cutaneous burns are also explained by the passage of toxic substances through the intestinal wall (Kijanitzin, "Virchow's Archiv," Bd. CXXXI). When these processes do not go far enough to cause the formation of intestinal ulcers, they may irritate the mucosa sufficiently to cause disturbances of secretion and resorption, which give rise to the symptomatology of dystrypsia already described. The treatment, wherever a treatment is possible, should be directed to the underlying cause. The details can be found in the chapter on Intestinal Ulcers, whenever the etiology points in that direction. The gastric and intestinal indigestions occurring in *gout* have a very manifold pathogenesis. From careful clinical study of many gouty subjects I have concluded that the intestinal dystrypsia is not caused by the gout so much as by the faulty diet and the medicaments with which gouty sufferers are apt to maltreat their digestive canal. As Watson aptly says, it is much more often a case of "*porck on the stomach than gout on the stomach*" (see also Grube, "Gichtische Erkrank. des Magens u. Darms," "Verhandl. d. XVIII. Cong. f. innere Med.," Wiesbaden, 1900, S. 195).

VIII. Dystrypsia Due to the Activity of Intestinal Parasites —Worms.

This has been described in all its details in chapter XXXIII on Intestinal Parasites.

IX. Dystrypsia Due to Hyper- or Hypoperistalsis.

The symptomatology, diagnosis, and treatment of these dystrypsias have been given in the chapter on Motor Neuroses of the Intestine.

General Considerations Concerning Intestinal Dystrypsia.

Whenever a dystrypsia has existed for a considerable time on a purely functional basis, it may eventuate in a disease with definite anatomical alterations. One of the most common symptoms is augmented intestinal peristalsis as a result of irritation by fermentative masses. A condition results in which the contents of the upper part of the bowel are hurried through the entire intestine in an unaltered condition—*i. e.*, the stools may present the same properties as are characteristic of the contents of the jejunum, which are normally thick, but liquid and gelatinous. The stools in the dystrypsias may frequently present the same characteristics —*i. e.*, they consist of feces mixed with unaltered food, and a large amount of glassy, frog-spawn-like mucus. In the chapter on Enteritis it has been emphasized that mucus is a sign of catarrh ; therefore the practitioner is called upon to distinguish the mucus in jejunal diarrhea from that passed in catarrhal inflammation of the bowel. The following features will serve for the differentiation. The jejunal stool is generally of an acid reaction, has only a slightly fecal odor, and is very rich in bile pigment, while the mucus in genuine catarrhal inflammation is not especially rich in bile pigments—frequently contains none at all, but contains epithelia and round cells, which are absent in the jejunal stools. The most helpful factor in the treatment is a carefully adapted diet. In adults who are otherwise fairly well nourished, total abstinence from food for forty-eight hours is the most effective means of treatment in the author's experience. Then the diet may be resumed and should be selected according to information given by gastric test-meals, and eventually duodenal test-meals and examination of the stools. When there is excessive bacterial activity, the various intestinal antiseptics have been advocated. Personally the author has been able to get along without them in the great majority of cases. He has seen very grave results follow the administration of the irritant antiseptics. Perhaps the most available are bismuth salicylate and subgallate, betanaphthol bismuth, thymol, menthol, and salol. Creasote, even in small doses, has, in the author's experience, deranged the stomach in sufferers from dystrypsias. In cases in which the gastric secretion of HCl was suppressed, he made a trial with the orexin so strongly recommended by Penzoldt, and noted in three cases an irritative diarrhea which became manifest after the first two doses, and ceased when the remedy was discontinued.

The following formulas have been used in a large number of cases by the author, for the special symptoms of intestinal dystrypsia. For the relief of constipation and diarrhea see the formulas given in the respective chapters on these subjects.

The following has proved useful in the putrefactive diarrheas, especially when associated with abdominal pain:

℞.	Tannigen, ...	4.00	gm.	(1 dram)
	Bismuth subgallate, ...	8.00	"	(2 drams)
	Salol, ...	1.55	"	(24 grains)
	Denarcotized extract of opium, ...	0.20	"	(3 grains).

This can either be made into twelve capsules or prescribed with six ounces of some elixir, of which the author prefers the elixir of gentian and the essence of calisaya (P. D. & Co.), three ounces of each, in doses of a tablespoonful three or four times a day.

The author's favorite recipe for anorexia from gastric hypochylia in intestinal dystrypsia is the following:

℞.	Strychnin sulph., ...	0.02	gm.	(⅓ grain)
	Dil. hydrochloric acid, ...	15.00	"	(½ ounce)
	Fluid ext. condurango, ...	45.00	"	(1½ ounces)
	Elixir of gentian, ...	180.00	"	(6 ounces).

M. SIG.—One-half of a fluidounce in two ounces of water, one-half hour before meals, through a glass tube.

Or:

℞.	Tinct. of nux vom., ...	9.30	gm.	(2½ drams)
	Essentiæ calisayæ (P. D. & Co.), . . .	60.00	"	(2 ounces)
	Elixir of gentian, ...	180.00	"	(6 ounces).

M. SIG.—One-half of a fluidounce thrice daily, one-half hour before meals.

When there are evidences of anemia with the gastric hypochylia, the following acts satisfactorily:

℞.	Quin. sulph., ...	1.16	gm.	(18 grains)
	Strychnin sulph., ...	0.02	"	(⅓ grain)
	Sulphate of iron, ...	0.80	"	(12 grains)
	Arsenious acid, ...	0.012	"	(⅕ grain).

M. SIG.—Make into twelve pills. One pill three times daily. They must be prepared fresh and not coated.

Boas uses the following powder for anorexia:

℞.	Ext. of nux vom., ...	0.024	gm.	(⅜ grain)
	Bismuth subcarbon., ...	0.520	"	(8 grains).

M. SIG.—Make twenty powders. One powder three times daily.

Menche has warmly recommended resorcin sublimate, and it undeniably improves the appetite in cases of incipient gastric and intestinal fermentation. It has also a slight sedative action. The following is Menche's formula:

R.	Resorcin. resub.,	2 gm.	(30 grains)
	Tinct. of rhubarb,	15 c.c.	(½ ounce)
	Simple elixir, enough to make	90 "	(3 ounces).
M.	SIG.—A tablespoonful twice daily.		

Resorcin can also be advantageously combined with bismuth salicylate, salol, and betanaphthol in the following manner:

R.	Resorcin resub.,	5 gm.	(75 grains)
	Bismuth salicylate,		
	Powdered rhubarb,		
	Sodium sulphate, of each,	10 "	(150 grains)
	Milk-sugar,	15 "	(225 grains).
M.	SIG.—An even teaspoonful twice daily.		

The following formulas are recommended by Ewald for anorexia with fermentation:

R.	Tinct. of nux vom.,	22.56 gm.	(6 drams)
	Resorcin. resub.,	5.40 "	(83 grains)
	Tinct. amar.,	11.25 "	(3 drams).
M.	SIG.—Take ten to fifteen drops every two hours.		

R.	Fluid ext. condurango,	17.00 gm.	(4½ drams)
	Resorcin. resub.,	4.00 "	(1 dram)
M.	SIG.—Thirty drops four times daily.		

CHAPTER XIX.

ENTERITIS (INTESTINAL CATARRH).

The following forms of enteritis are distinguishable as morbid entities and merit separate consideration :

1. Acute simple enteritis.
2. Chronic simple enteritis. (These two are often spoken of as simple acute and chronic intestinal catarrh.)
3. Phlegmonous or purulent enteritis.
4. Diphtheric or croupous (pseudomembranous) enteritis.
5. Membranous or mucous enteritis (colica mucosa et enteritis membranacea).

In addition to the foregoing, Maragliano ("Berlin. klin. Wochenschr.," 1894, No. 13) has described a special ulcerative enteritis of severe type which is almost exclusively limited to the ileum, and in which a type of the colon bacillus was demonstrated as the probable exciting cause. The disease began with a prodromal stage, in which there was no fever, but constipation and abdominal pains. Then followed the clinical picture of typical enteritis, with strong meteorism, constipation, diarrhea, enteralgia, and fever. The cases eventuated into perforation peritonitis, with symptoms of intestinal obstruction. The process, so far as has been observed, begins with hemorrhagic foci between the peritoneum and the mucosa, which may result in ulcerative destruction of the mucosa, serosa, or the entire intestinal wall. More accurate and detailed knowledge concerning this very intense, peracute enteritis has as yet not been obtained.

1. ACUTE ENTERITIS (ACUTE INTESTINAL CATARRH; ENTERITIS ACUTA).

Nature and Conception.

The various acute inflammations of portions of or the entire small intestine constitute the most frequent diseases of the digestive tract.

They may occur at any age, but they are most frequently observed in young children of the poorer classes, as a consequence of unsuitable nutrition, very often resulting fatally in these cases, especially when the child has already been weakened by other diseases.

The disease appears either as a primary or independent disease—idiopathic enteritis—or, secondarily, as a result of numerous other diseases—secondary symptomatic enteritis.

The former is most frequently produced by improper diet, such as the eating of unripe fruit, meats and fish which have commenced to decompose, the drinking of foul water, and also by foreign bodies, among which are to be mentioned biliary and fecal concretions, and also intestinal parasites. In addition, severe colds and traumatism may give rise to acute enteritis. This secondary form may appear in conjunction with almost all diseases, especially in conjunction with certain acute infectious diseases; also with nephritis, diseases of the lungs, the heart, and the blood-vessels. It may accompany typhoid, syphilitic, and tuberculous ulcers. The anatomical changes in acute enteritis, which consist of intense hyperemia, swelling of the intestinal mucosa, which may sometimes lead even to superficial loss of substance, are occasionally restricted to only a portion of the mucous membrane of the intestines; thus we have duodenitis, jejunitis, ileitis, colitis, and proctitis. But more frequently they extend over a large area of the intestinal mucosa, especially the large intestine; less frequently of the small intestine, but very frequently over both the large and the small intestine. In addition to the changes mentioned, there is a more or less copious production of mucus. The most important symptoms of the disease consist of loss of appetite, inclination to vomiting, especially when the stomach is also affected, violent rumbling in the intestines, tenesmus, more or less violent pains in various portions of the abdomen, according to the localization of the disease, but often referred to the umbilicus, thin, liquid evacuations, the frequency of which is dependent upon the character and extent of the anatomical changes, and, in certain cases, fever, which, especially in children, may run very high.

If numerous new injurious influences act upon the inflamed mucous membrane during the continuance of the disease, the inflammation may pass over into a chronic state. At certain seasons of the year, especially in summer, when more injurious influences act upon the intestinal mucosa than at other seasons, acute enteritis often appears epidemically.

Adherence to correct diet and rest and avoidance of injurious influences will effect a cessation of the enteritis in most cases, but in convalescents from other diseases or cachectic, ill-fed children less capable of resistance, enteritis may result in death, especially with unsuitable treatment and diet.

Etiology.

Most all forms of enteritis may be classified etiologically into two groups: (1) Those forms caused by pathogenic micro-organisms, either by direct invasion or by the effect of toxins generated by them. These may be designated as the "*infectious catarrhs.*" (2) Those forms caused by chemical poisons, which may act upon the intestines from three different sources: (*a*) They may be introduced with the ingesta; (*b*) they may develop from the ingesta through putrefaction; (*c*) they may act from the blood. All the forms of the second group are designated as "*intoxicational catarrhs.*"

Various other Causes.—Enteritis can undoubtedly be brought on by cold; this "enteritis rheumatica" with diarrhea is in all probability due to pathological alterations characteristic of catarrh. Mechanical irritations and severe traumatism to the external abdomen are supposed to be sufficient to cause the disease, though this is hypothetical; internal irritants of a mechanical nature arising within the bowel certainly do cause catarrh. It is known that gall-stones, foreign bodies, and hardened fecal masses act in this manner (see section on constipation and ulcerations arising therefrom). A secondary catarrh is usually found in the neighborhood of carcinoma, intestinal constrictions, and occlusions, and coincident with some forms of peritonitis. The various entozoa are supposed to be causes of enteritis, acting either directly or through chemical toxins produced by them (see chapter on Intestinal Parasites).

Primary and idiopathic acute intestinal catarrh as an independent disease is frequently caused by errors of diet, by food or drink that is qualitatively or quantitatively abnormal. In case abnormal quantities of food and drink are taken into the intestinal canal, the walls of the intestines may be distended excessively, and thereby irritated mechanically, or the ingesta may remain longer than usual in the small intestine, since the secretions do not suffice to digest them. In this case the ingesta may become subject to putrefaction, the products of which irritate the mucous membrane chemically, causing inflammation. This is liable to happen especially when the irritability of the mucous membrane has been materially increased by previous diseases or by disturbances of circulation. Furthermore, acute enteritis arises very frequently after eating spoiled meats, fish, oysters, unripe fruit, potatoes, or after drinking sour, decomposed milk, beer which is not seasoned, foul water, etc.

There is no doubt that severe inflammations of the intestinal mucous membrane are sometimes caused by severe drastic purgatives—for instance, colocynth and croton oil; also, poisons and corrosive substances, taken either accidentally or intentionally, such as arsenic, corrosive sublimate, carbolic acid, unusual doses of antimonium and potassium tartrate, caustic alkalis, as well as organic (carbolic acid) and inorganic acids (HNO_3, HCL, H_2, SO_4).

The increased heat of summer causes more decomposition and fermentation of foods, and the ingestion of large quantities of cold liquids, conditions sufficiently explaining the increased frequency of enteritis at this season. Foods containing large quantities of cellulose dissolve very slowly in the gastro-intestinal canal, and, irritating the mucous membrane of the intestines more severely, may cause acute enteritis.

Foreign bodies—coins, little stones, bits of wood or bone, fishbones, fruit-seeds, also gall-stones, fecal concretions—and, in addition, the larger intestinal parasites—teniæ, ascarides, round worms, oxyures, etc.—all come under consideration as mechanical irritants.

Enteritis After Extensive Cutaneous Burns.—Hunter was among the first to suggest that toxic substances, formed of charred skin and broken-down blood constituents in the charred skin, becoming absorbed into the circulation, were later excreted with the bile into the small intestine, there exerting a deleterious influence on the mucosa.

Kijanitzin claims to have isolated a ptomain from the blood and organs of animals with extensive cutaneous burns. This substance, when injected into other animals, produced the symptoms usually observed after large burns, . . . among which were diarrhea and passage of blood. It is not stated whether any lesions of the duodenum are produced thereby (Kijanitzin, "Virchow's Archiv," Bd. cxxxi, S. 436).

Colds.—A very cold drink may be followed by diarrhea, the most frequent symptom of acute enteritis, but this diarrhea is generally only the result of a temporary increase of peristalsis, which must not be identified with acute enteritis.

After sudden severe drenching and chilling of the freely perspiring body-surface acute catarrh of the intestines may set in. The pathological physiology of the latter has not been cleared up, but it is probable that disturbances of the circulation in the intestinal vessels, together with the nervous influences, play an important rôle.

Infectious Enteritis.—There are two essential groups under this type: (1) Those in which the pathogenic action of the micro-organisms is limited to the gastric intestinal canal. (2) Those in which the micro-organisms have invaded other portions of the organism and the intestinal affection is only a partial localization of a general infection.

The bacteria that exert their influences exclusively on the gastro-intestinal canal may set up enteritis directly or indirectly. All bacteria capable of generating poisonous, chemical irritants from intestinal contents can produce this effect; it is therefore the effect of the indirect action of obligate intestinal parasites. From observations made up to the present time it seems probable that catarrhal and other inflammatory diseases of the intestines are not produced by specific constantly recurring microbes, but that many kinds of bacteria are capable of producing these effects, under varying conditions which are thus far not thoroughly understood.

Gărtner was first to attempt demonstration of an organism which is directly pathogenic—*i. e.*, producing enteritis directly. In the flesh of a cow exhibiting signs of enteritis and also in the spleen of a man who died twenty-four hours after ingestion of this meat he found a characteristic organism which, when injected into or fed to mice and other animals, proved poisonous and causative of enteritis. He gave it the name of "Bacillus enteritidis." These organisms are short, movable rods, occurring frequently in pairs, and surrounded by small circular areas; one pole stains deeper than the rest of the rod. They grow on gelatin without liquefying it. In stab culture they grow only slightly downward, but move on the surface as a thick, grayish-white coating. On agar, potato, and blood-serum, they grow rapidly but form no spores. In the culture-medium the organism produces toxins. Lubarsch confirmed the existence of the same bacillus in a case of septic pneumonia occurring in a neonat. Nothnagel considers, on the evidence of Gärtner, that there are organisms that produce enteritis directly (*l. c.*, p. 97). It is, however, probable that the Bacillus enteritidis of Gärtner is identical with one of the types of colon bacillus.

Cohnheim was one of the first to assert that some of the cases of intestinal catarrh which occur epidemically in summer are of infectious origin. It is probable that in the case of acute infectious catarrh of the intestines the inflammation of the mucous membrane is caused not so much by the micro-organisms themselves, as by their poisonous metabolic products, which, after being absorbed into the blood, effect an increased peristalsis, the main cause of diarrhea. Recently the view has been gaining ground that the forms of catarrhal enteritis are not traceable to specific, constantly recurring pathogenic microbes, but to the commonly present obligate or occasional bacteria, which become virulent, and thereby obtain the power to produce those diseases.

Herbert E. Durham has reported an epidemic of enteritis involving about 185 persons, in which the specific organism appears to have been the Bacillus enteritidis ("Brit. Med. Jour.," September 3, 1898). In 29 cases the serum of individuals who had recovered from the disease caused agglutination in dilutions of 1 : 100 or higher of a culture obtained from the liver of a fatal case. Of 8 samples of serum from convalescents, 6 had marked protective and agglutinative action and also gave Pfeiffer's test.

Secondary or symptomatic enteritis is a term applied to intestinal catarrhs occurring as a sequence to diseases of other organs, more especially to an extension of the disease from the stomach or the rectum. Any one of the various forms of gastritis may spread over the mucous membrane of the intestines by putrefactive products contained in the chyme, or by direct cellular extension of the inflammatory process. Catarrh of the rectum may extend to the lower portion of the large intestine. In women with gonorrhea a large quantity of secretion may be carried into the anus and there cause inflammation. (Concerning the various types of proctitis and their extension see chapter on Diseases of the Rectum.)

Enteritis Accompanying or Following Infectious Diseases.—During epidemics of typhus, dysentery, and cholera, acute enteritis also occurs with great frequency, and is considered as a milder form of the infectious process. It is assumed in these cases that the specific pathogenic organisms invaded the system only in attenuated form. Acute intestinal catarrh has repeatedly been observed in malarial infections; in these cases the symptoms can quickly be removed by quinin, which suggests in these cases a localization of the malarial parasite in the mucous membrane of the intestines. Secondary catarrh of the intestines may appear in the course of other febrile diseases. In these cases we should recall that, with high fever, the production of hydrochloric acid in the gastric juice is much diminished or suppressed entirely, and the gastric peristalsis reduced; the consequent stagnation and indigestion lead to decomposition of the chyme, which is converted into a putrefying mass, a source of infection to the intestinal mucosa—especially so when intestinal peristalsis is also impeded (Hans Herz, "Störungen d. Verdauungs-Apparates, u.s.w.," S. 118).

Acute catarrh of the intestines often appears in the neighborhood of well-marked pathological lesions, such as tuberculous, syphilitic, typhoid intestinal ulcers, carcinoma, and stenosis of the intestines. Incarcerated herniæ and volvulus and intestinal invagination will generally be associated with an acute enteritis. Amyloid degeneration of the mucous membrane of the intestines may complicate acute enteritis. Disturbances of the circulation of the intestinal walls, and especially of the mucous membrane, favor the appearance of this disease, especially in those cases in which the venous hyperemia causes a lowering of peristalsis. But it can not, even after it has reached an exaggerated degree, cause acute enteritis directly. The accumulation of the feces in the intestine constitutes a mechanical and often a chemical irritation to the mucosa, and mostly stained with blood. In very severe cases also the upper layers of the submucosa are richer in blood than normally.

Swelling of the Mucous Membrane.—This is manifested by loosening, greater susceptibility to laceration, and increase in thickness; the latter is dependent on an inflammatory edema or merely serous infiltration. In severe cases, especially with secondary catarrh, the submucous tissue is also affected by the swelling.

The intestinal epithelia are partially cloudy, affected by fatty and granular degeneration. Extensive separation of the epithelial integument, especially if tissue fragments are mixed with the contents of the intestines or are lodged in the mucous membrane of the intestines, is a result of postmortem change.

But that an abnormal desquamation of epithelia also takes place during life is proved by the greater abundance of cells in the dejections at the same time. In the case of idiopathic catarrh, the solitary follicles are more or less affected by the inflammation, and in secondary catarrhal inflammation, also the Peyer's patches. At the beginning the enlarged follicles project as grayish, translucent knots above the level of the mucous membrane (inflammatory edema).

As the acute enteritis continues, a copious increase of the specific tissue elements and an immigration of white blood-corpuscles become manifest, the follicles now presenting a grayish-white appearance. With very rapid increase of the cells necrosis may ensue, the follicles burst, and a follicular ulcer be formed (enteritis follicularis sive nodularis).

Follicular ulcers, which are generally observed only in the case of severe catarrh, must be distinguished from catarrhal ulcers, which probably arise from sequestration of superficial epithelium (catarrhal erosions), which, under the influence of mechanical and chemical irritation, develop into ulcers.

Increased Secretion.—The mucous membrane in acute enteritis is, in less severe cases, covered with a transparent, tough, glassy mucus which may be more or less colored by bile or blood.

With more intense inflammation the mucus becomes opaque, due to an admixture of epithelia and leukocytes. If the irritant which acted upon the mucous membrane be stronger, the secretion becomes purulent.

The muscularis and serosa of the intestine are generally unaffected; the *mesenteric glands*, especially in secondary catarrh, are enlarged, succulent, and hyperemic. Concerning the contents of the intestines and the character of the dejections, see the Examination of the Feces and Symptomatology.

Pathological Histology of Acute Enteritis.

The most characteristic signs of an acute catarrh are an abundant accumulation of round cells in the upper portion of the submucosa, along the muscularis mucosæ, and also around the blood-vessels, distention of the vessels of the mucosa and submucosa, small extravasations between the glands of Lieberkühn, hyperplasia and swelling of the solitary follicles and Peyer's patches. The epithelium of the mucosa in the colon is always detached, but this is usually regarded as a postmortem change, because it has been observed in almost every cadaver that comes to the autopsy table later than six hours after death. The epithelium of the intestinal mucosa no doubt takes part in the general inflammatory process. The epithelium of the glands is often unchanged, but generally appears cloudy and swollen. The glands of Lieberkühn are changed in form and position. In localities they appear loosened, displaced, and in part pushed into the lumen of the intestine or entirely disrupted from their normal position. Wm. G. Booker ("Johns Hopkins Hospital Reports," vol. VI), in a study of the gastro-enteritis of children, found that in most cases there was a loss of epithelium over some parts of the surface. In a few cases it was almost entirely lost over the whole intestinal surface. Where the epithelial layer was preserved it was infiltrated with mononuclear and polynuclear leukocytes. Booker took every precaution for thorough preservation of his tissues, placing them in preserving fluids within a few minutes after death. They may, therefore, be safely said to represent conditions that existed during life. Nevertheless, he found necrosis of the mucosa in some of his cases. The glands of Lieberkühn are, as a rule, expanded or dilated, this dilation being either uniform or occurring in the fundus alone, giving the gland a bottle-shaped appearance, owing to the constriction near the orifice. These histological changes pave the way for the more penetrating destructions of the parenchyma—the catarrhal and follicular ulcers. It is evident, however, that bacterial and toxic agents must act simultaneously with the changes above described.

Symptomatology.

The clinical picture of acute enteritis varies considerably according to the location, the extent, the intensity, and the cause of the disease. In primary idiopathic acute enteritis the symptoms of this malady only are observed; in secondary catarrh the symptoms of the primary disease are also associated, and the aspect of the disease becomes more manifold. Acute enteritis as an effect and partial phenomenon of other diseases is discussed together with these in another chapter. In the following only the symptoms of idiopathic acute enteritis will be portrayed, and especially those of acute catarrh of the jejunum, ileum, and colon, which most frequently occur together. In addition to these, the symptoms of catarrh which is restricted to smaller portions of the intestines (duodenitis, colitis, proctitis) will afterward be discussed in so far as they can be delimited.

Primary Idiopathic Enteritis.—This generally arises very suddenly,—soon after the action of those injurious elements which have produced it,—manifesting itself in diarrhea. More rarely this is preceded for a short time by feelings of pressure and fullness in the abdomen, as well as by griping or colicky pains which become less violent after the first dejection, but in severe cases they afterward increase again.

Diarrhea is a very frequent, but not at all constant, symptom of acute enteritis, since it may be wanting in cases where the duodenum, jejunum, or ileum, the whole small intestine, or even the latter in addition to the ascending colon, are alone affected. Diarrhea is absent when the large intestine, or the larger portion of the same, functions in a normal manner; the retardation in the transverse and descending colon may fully compensate for the increased peristalsis of the small intestine. If, in the case of isolated intestinal catarrh, which, according to the comprehensive observations of Woodward,* is exceedingly rare, thin, liquid evacuations of the bowels take place, we are to infer that increased contractions of the musculature of the large intestine have been caused by the irritating liquid chyme on its entrance into the colon, especially in the beginning of the disease. This irritation, however, does not go far enough to turn into catarrhal inflammation of the mucous membrane. On the other hand, in the case of extensive acute catarrh of both the small and large intestines, with increased peristalsis of the latter, the appearance of liquid dejections is always to be expected. If they do not appear, we are to assume that a paralysis of the muscular apparatus of the intestines is responsible for it, which, it is true, is only exceptional with acute enteritis or gastro-enteritis, but which is more frequently observed with Asiatic cholera and cholera nostras.

---

* Woodward, "The Medical and Surgical History of the War of the Rebellion," vol. 1, p. 2, 1879.

In acute enteritis the diarrhea is caused chiefly by an abnormal increase of the peristalsis, which causes the chyme and feces to be hurried on and out so quickly that the greater part of the water taken in with the food and the greater part of the gastric and intestinal juices are not absorbed. Further causes of diarrhea are the diminution of absorption, caused by disturbance of circulation, and also a copious collection of mucus, as well as of serous transudation, into the intestinal lumen. This explains the fact that in rather severe cases of acute enteritis the quantity of the liquid evacuated in the dejection may be larger than that which was introduced in the food and drink. The number of diarrheic evacuations varies considerably according to the intensity and extent of the disease—in light cases there may be only two or three, in severe cases fifteen or twenty, in twenty-four hours; rarely a greater number. If the colon at the beginning of the disease contains fecal matter which has already become thickened, this will be evacuated first in a thick, pulpy state; the following dejections are thin, pulpy, and finally they become liquid. If, however, the colon at the beginning of acute enteritis is empty or nearly empty, then the evacuations are liquid from the start. While the first dejections are generally colored brown and still contain rather large quantities of feces of a very penetrating odor, the subsequent evacuations, in case they follow in quick succession, are colored pale yellow and consist mostly of secretions of the gastro-intestinal canal, mucus, and serum.

In cases of simultaneous catarrh of the small and large intestines, Gmelin's reaction for unchanged bile pigment gives a positive result, which is the more frequent the further up the catarrhal inflammation of the small intestine extends, and the more the peristalsis of the intestines has been increased. Even in isolated catarrh of the small intestine unchanged bile coloring-matter may sometimes be demonstrated if the peristalsis of the colon is increased. The particles of mucus, epithelia, and white blood-corpuscles which are mixed in with the dejections are also often tinted by unchanged bile pigment (microchemical reaction). If the catarrh is confined to the small intestine, and if the peristalsis of the colon is normal, or if only the latter is affected, unchanged bile pigment is lacking in the evacuations. The presence of bile pigment in the discharges depends upon the rate of peristalsis.

We are justified in assigning the origin of bile-stained mucus to the jejunum and upper ileum only; if it consists of small particles, just recognizable by the naked eye, which are intimately mixed with the food particles, and if the mucus shows crystals or granules of bilirubin or partially digested cells under the microscope.

The simple presence of bile-stained particles in the feces does not prove the existence of an enteritis in the small intestine. It requires the demonstration of remnants of muscle-fibers, starch granules, excessive primary fermentation of the feces, and the above-mentioned particles of mucus to establish involvement of the upper small bowel (see Chap. IX).

The pale yellow diarrheic dejections of adults sometimes assume a green color after the disease has progressed; in case of small children, they are sometimes colored green from the beginning, and in that case generally give a very pronounced bile reaction. If the diarrheic evacuations follow in quick succession, they have sometimes the appearance of the clear, watery evacuations which are characteristic of Asiatic cholera and also of cholera nostras. Often they contain larger or smaller hard scybala, and clear watery evacuations may alternate with fecal passages. In case of catarrh of the small, as well as of the large, intestine, the dejections always contain mucus, which can be demonstrated macroscopically and microscopically; it appears in larger or smaller quantities, and either intimately mixed with the thick or thin, pulpy feces (catarrh of the small intestine) or in the form of pieces, shreds, or lumps of mucus, balls of mucus, and yellow-colored grains of mucus.

In the microscopic examination of the dejections of acute enteritis, well preserved or more or less altered (flaky) epithelium and white blood-corpuscles are found. If the hyperemia and inflammation have reached an advanced degree, or if ulcers have been formed, one finds small masses of blood,—rarely, large ones,—red blood-cells more or less disintegrated, and perhaps hematin crystals. With acute catarrh of the lowest portion of the intestine (colon and rectum) the scybala formed are surrounded by a layer of mucus more or less thick, which often has a bloody tinge, or else pure mucus is evacuated alternately with the feces. The quantity of undigested or half-digested matter in the evacuations is mainly dependent upon the location and extent of the disease. In case of catarrh of the small and of the large intestine, with increased peristalsis of the latter, the quantity of undigested food particles in the dejections is largest, especially when much food has been taken in. Muscle-fibers, albuminous particles, lumps of casein, and large quantities of undigested fat are met with. If the evacuations contain well-preserved starch particles in large quantities, it points to very defective or inhibited secretion of pancreatic juice, or it is a consequence of highly increased peristalsis or decreased absorption. In catarrh of the colon alone, in case the small intestine functions normally, the proportion of undigested matter in the evacuations is little or not at all increased, since upon the entrance of the feces into the large intestine the digestion and absorption of the food have been almost completed (see chapters on Macroscopic and Microscopic Examination of the Feces and on Motility or Peristalsis of the Intestines). In exceptional cases diarrhea may be the only symptom of acute enteritis, but then the diagnosis is to be made with caution, in order to avoid confusion with nervous diarrhea. If the dejections contain large quantities of air or gases, they are foamy. During the evacuation, or even without evacuation, more or less offensive flatus is emitted. The evacuation of very liquid stools is occasionally involuntary.

The emission of flatus may be caused by an abundant collection of gases in the intestine, in consequence of strong fermentation and putrefaction, but this is not always the case. Under normal conditions air and gases often remain for some time in the intestines, and a part of the same is furthermore quickly absorbed. In acute enteritis in consequence of increased peristalsis a small accumulation of gases will often suffice to stimulate peristaltic contractions.

Subjective Complaints.—Unpleasant sensations of pressure and fullness in the abdomen, griping as well as colicky pains, are the main subjective symptoms. The diarrhea and feeling of fullness are generally the result of the inflammation itself; the others are due to spastic contractions of the muscular system of separate parts of the intestines. The griping is generally increased, the colic diminished, by pressure. The localization of the pains gives no sure indication of the location of the disease, since they often radiate in various directions, and since, on account of the great movability of the intestinal loops, reliable localization is hardly possible. Only when they can be shown to be present continuously in the course of the transverse, ascending, or descending colon, or when they are confined to the right iliac region, are we to infer disease of the portions of the intestines in question. The sensitiveness to pressure of the abdomen often continues for some time after the catarrh has ceased. In catarrh of the rectum and of the lowest part of the large intestine patients are especially troubled with a very torturing tenesmus, which, in severe cases, compels them to remain at stool continuously. In acute intestinal catarrh prolonged rumbling and gurgling sounds are heard, sometimes audible at some distance from the body.

The result of the physical examination is different in the various cases. Sometimes the whole abdomen is distended meteoristically, and the patients then complain, on account of the pressure on the diaphragm, of want of breath, palpitation, precordial fear, and asthmatic complaints. More frequently the abdomen is arched only irregularly, especially in the region of the transverse colon. The increased peristaltic movements of the intestines are at times visible, and on palpating the abdomen one sometimes feels a distinct gurgling (ileocecal noise). If the intestines contain, at various places, large quantities of air and liquids, one notices a fluctuating feeling, which may be accompanied by distinctly audible splashing. Percussion, of course, gives entirely different results, according as the various parts of the intestines contain air or liquids. The results of percussion can not be used for the localization of the disease.

Derangements on the Part of the Stomach.—In acute enteritis the appetite is diminished or complete anorexia exists. Nausea, severe vomiting, tenderness in the region of the stomach, coated tongue, and pasty taste may be present. If the acute enteritis has arisen from the inflammation of gastric mucosa that has extended to the intestinal mucosa, which is especially frequent in children, then the symptoms mentioned appear sooner than those of acute intestinal catarrh. The vomiting is at times so persistent as to interfere seriously with the giving of medicines by the mouth. On the other hand, although this is rarer, the inflammation of the mucous membranes of the intestines may extend secondarily to the stomach; or, *e.g.*, mistakes of diet cause simultaneous gastritis and enteritis. Furthermore, the derangements on the part of the stomach are not always to be referred to gastritis, since they may arise in a purely reflex manner. With catarrh of the large intestine and of the rectum the appetite is generally not at all diminished.

Condition of the Kidneys and of the Urine.—When the loss of water through the intestine has been considerable, oliguria is observed. This oliguria is occasionally seen as a result of stubborn vomiting; sometimes, also, after temporary anorexia. The *urine* is concentrated and dark, and on standing for some time often deposits an abundant sediment, consisting of urates. With catarrh of the small intestine it often contains large quantities of indican, which is an indication of excessive intestinal putrefaction. The methods of testing for indican are given in chapter IX.

In cases of acute enteritis associated with fever, but sometimes also in afebrile cases, hyaline casts, either with or without albumin, can be found in the urine (Fischl), often only a few hours after the beginning of the disease, especially when the discharges are very copious. Besides the casts, the urine contains renal epithelia and white blood-corpuscles; seldom, red cells. According to Stiller, a slight albuminuria, in addition to the cylindruria, is a frequent partial symptom of simple acute gastro-intestinal catarrh (see Chap. IX).

Fever.—This is absent entirely in most cases of acute enteritis, especially with adults. The disease may, however, commence with repeated chills, even very severe ones. If the increase in temperature remains for several days at about 112° F., we may suspect a case of enteric fever, especially when the acute enteritis causes swelling of the spleen (Fischl). The result of the Widal test, careful measurements of temperature (in case of typhoid fever gradual increase of the fever), the nature of the pulse, which, in feverish acute enteritis generally rises quickly, the absence of typhoid spots, guard against mistaken diagnosis. In the feverish cases the rise in temperature is very probably the result of an infection through pathogenic micro-organisms. With small children, irritable, as well as feeble, old people, the fever may be high (105° F.) and lead to delirium and convulsions.

The general health is, in light cases, very little disturbed, but in severe cases it is seriously damaged. Patients feel as weak and weary, in some cases, as if a severe sickness were coming upon them. In small children and weak old persons the signs of a severe collapse sometimes appear. The eyes are sunken into the head, the facial expression is that of suffering, the skin is sometimes covered with cold sweat, the turgor of the same decreases, the lips are bluish, and the extremities are cool. The symptoms of acute hydrocephaloid disease, described by Marshall-Hall, which has been attributed to anemia of the brain in consequence of large loss of water, are observed in rare cases.

Generally, hydrocephaloid disease is a result of cholera nostras (diarrhea with vomiting), and is accompanied by very high fever—the temperature may then rise to 104° F. Generally a strong, rather sudden collapse sets in then, and the extremities are cold while the body is hot, the fontanels sink in, the pulse is considerably accelerated, very weak, and often irregular. Dyspnea, convulsions, delirium, and stiffness of the neck also set in. The children are in an apathetic state, and cry out suddenly only when more severe colicky pains seize them. Very often the little patients perish in a short time in consequence of paralysis of the heart; bronchopneumonia and thrombosis of vessels may also set in as complications. In isolated cases of severe enteritis intense pains in muscles and joints, even slight swellings of joints, are observed; in these cases we probably have to deal with infectious enteritis.

Course and Extent of the Disease.

With proper care and the avoidance of injurious ingesta complete recovery may result in a few days in light cases. But in severe cases the duration of the disease is somewhat longer, and improvement and relapse may alternate, especially when the patient does not take proper care of himself. After the setting in of convalescence, the intestines are frequently very sensitive to injurious matter, even for a considerable time. If no regard is paid to this, acute enteritis with frequent relapses may turn into the chronic state. Frequently, in consequence of great exhaustion of the muscles of the intestines, constipation may set in, which departs again after a time, but which may also give rise to new diseases. The greatest care is to be exercised in order to avoid the acute condition becoming chronic.

Localization of the Catarrh.

Catarrh of the Duodenum.—If the catarrhal inflammation is restricted to the duodenum, it may be recognized only when icterus results, in consequence of the clogging up of the ductus choledochus or the swelling of its mucous membrane. In the cases where the latter is absent, in spite of catarrh of the duodenum a sure diagnosis is impossible.

If icterus is absent, catarrh of the duodenum may be inferred only then when, in conjunction with acute gastritis, a constant increased sensitiveness appears in the right hypochondrium and mucus is found macroscopically and microscopically in the evacuations. Duodenitis is most certainly to be diagnosed, even when icterus is absent, when the above-mentioned symptom appears after extensive skin-burns, since Hunter has suggested that the poisonous matter which there arises is secreted by the bile into the intestines, and may cause a severe inflammation of the mucous membrane of the duodenum, which may result in the formation of ulcers or even in perforation of the intestinal walls.

An isolated catarrh of the jejunum or of the ileum can not be diagnosed during life. The detection of simultaneous diseases of the jejunum and ileum, even if present, is difficult to diagnose, especially as the most important symptom of catarrh, the diarrhea, may be absent. Only by a thorough microscopic examination of the feces may data be gained for the demonstration of isolated catarrh of the small intestine (Nothnagel). The mucus in the same is increased and intimately mixed with the feces, and in addition one sometimes finds in the feces yellow grains of mucus of the size of a poppy-seed. If the peristalsis of the large intestine is not at the same time increased, the yellow grains of mucus, as well as the yellow-colored white blood-corpuscles, will not give the microchemical reaction for unchanged bile pigments. Sometimes the increase of undigested particles of food, especially starch, in the dejections can be utilized in the diagnosis. The amount of indican also, as has been noted, is frequently increased with catarrh of the small intestine.

Even in simultaneous catarrh of the small intestine and ascending colon diarrhea may be absent. For the rest, the results of the examination of the evacuations are about the same as in isolated catarrh of the small intestine.

Acute colitis, in contradistinction to the diseases mentioned above, is characterized practically always by the presence of liquid evacuations. In these are found larger or smaller masses of mucus which, like the feces, may contain bile pigment in case the peristalsis of the small intestine is at the same time increased. In acute colitis the pains and sensations of pressure which occur are restricted to the colon, and on palpation audible rumbling and splashing noises are elicited.

Inflammation of the Lowest Portion of the Intestines (Proctitis).—The characteristic symptom of proctitis is tenesmus, with severe antecedent colicky pains in the left iliac fossa. Often the patients feel the necessity of evacuation, but then each time only very small quantities of feces are passed. The evacuation also is frequently accompanied by severe pains, and the left iliac fossa is sensitive to pressure. The scybala are surrounded by a layer of mucus of greater or less thickness, which is often tinted with blood. Between these, after severe straining, quantities of pure mucus may be evacuated. Often, in defecation, a portion of the rectal mucosa protrudes from the anus in the shape of a dark-red roll. Spasmodic convulsions of the anus can often be demonstrated, and digital examination produces severe pain. Sometimes the finger is held spasmodically by the sphincter. The mucous membrane of the rectum feels hot, is slippery, and the finger, after being pulled out, is often covered with mucus. It was previously noted that with the occasional paralysis of the sphincter, mucus in large quantities passes off involuntarily, and inflammation may arise in the vicinity of the anus. With the characteristic symptoms of acute proctitis a digital examination or the use of the rectal mirror is generally unnecessary.

Clinical investigations tending to localize the intestinal catarrh are of decided practical interest, because the treatment and methods employed must vary in individual cases with regard to the intestinal district that is preeminently or exclusively affected. The most frequent locality that is affected is the colon, but the other variations are the following: (1) Enteritis involving the entire small intestine and the entire colon; (2) the colon only; (3) the small intestine exclusively; (4) the beginning of the small intestine, the duodenum, and the upper portion of the jejunum; this is most frequently a result of, or in connection with, gastritis; (5) the end of the large intestine—proctitis; (6) appendicitis; (7) enteritis affecting only the cecum and the ascending and the transverse colon; (8) the disease affecting these latter portions and, in addition, the lower part of the ileum; (9) Mayor ("Quelques mots sur une variete d'enterite iliaque," "Revue medicine de Suisse romande," 1893) has described an inflammatory catarrh, affecting exclusively the sigmoid colon, under the name of *sigmoiditis*. The clinical and anatomical picture of this disease is said to correspond closely with that of typhlitis.

The results of auscultation, percussion, and palpation for the localization of enteritis are unreliable. Pain, if sharply circumscribed, may be available for the purpose of localizing the enteritis. Two kinds of pain are to be distinguished for this purpose: that which arises spontaneously and that which becomes evident only on pressure. The following are the few characteristic pains which are significant: that localized in the right iliac region in typhlitis and appendicitis, the tenesmus which is characteristic for proctitis, and the sensitiveness to pressure along the anatomical course of the colon. It is always necessary to examine the entire rectum and sigmoid flexure by the long rectoscope and sigmoidoscope if the patient can stand it, for in acute proctitis it is often too painful. Inflammatory catarrh affecting these portions, and even affecting the descending colon, can be diagnosed in this manner by direct inspection. The indications that may be derived from the condition of the urine and examination of the dejecta have been considered in a separate chapter.

For the sake of context I will repeat here that the evacuation of pure mucus without any admixture of fecal matter whatever indicates proctitis—catarrh of the sigmoid or descending colon.

Very small balls of fecal matter completely enveloped in mucus point to the same locality.

Hyaline clumps of mucus that are recognizable by the microscope only and intimately mixed with the firm or semisoft excrement are suggestive of catarrh of the cecum and of the ascending and the first part of the transverse colon. This microscopic condition is observed also where the catarrh exists in the lower part of the ileum.

A very intimate commingling of mucus and excrement, no matter whether the latter is firm, semisolid, or liquid, is, as a rule, an indication of catarrhal involvement of the higher sections of the colon or lower part of the ileum.

A catarrhal affection of the entire colon is accompanied by a thin dejection, showing very intimate admixture of small fragments of mucus, which are recognizable by the naked eye. When the catarrh is restricted to the descending colon and rectum, there is no such intimate admixture of mucus and excrement.

Small granules of mucus stained intensely yellow have already been described as suggesting an affection of the small intestine. Concerning the reaction of bile pigments, we may summarize the following: A stool may give the bile pigment reaction throughout, or the pigment may be attached to individual constituents, especially to the particles of mucus in it. A reaction that occurs in this manner suggests catarrhal inflammation of the entire colon, involving simultaneously the lower portions of the ileum. The more pronounced the reaction, the higher up into the ileum does the catarrh extend. Cylindrical epithelial cells, round cells, or fat droplets stained with bile point to involvement of the ileum, and perhaps also of the jejunum and duodenum.

The nature and quantity of undigested food particles give only uncertain information concerning the involvement of the small intestine. It is generally conceded, however, that an abundance of undigested muscle-fibers, in the absence of fever, indicates an involvement of the small intestine. In febrile conditions muscle-fibers appear undigested even in the absence of catarrh of the small intestine. An abundance of undigested starch in the stool in the presence of other symptoms of enteritis is a sign of involvement of the small intestine. It must be borne in mind, however, that the various forms of intestinal indigestion may produce the identical signs, even in the absence of enteritis, especially when the peristalsis is increased. For more exact diagnostic deductions to be drawn from the condition of the stools reference must be made to chapter IX on Examination of the Feces.

Diagnosis.

The recognition of acute enteritis is difficult when either the characteristic symptom, diarrhea, is absent, or when this is the only symptom of the catarrh. In both cases it is only after careful examination of the evacuations that a sure diagnosis can be made or confusion with stercoral, dyspeptic, or nervous diarrhea avoided. The most important thing is to note whether mucus is present in the dejections and can be demonstrated in greater or smaller quantities macroscopically and microscopically. In the above-mentioned forms of diarrhea this is absent. The presence of uncombined bile coloring-matter is in itself no sure criterion of catarrh of the small and the large intestine, since it is found also in the dejections when marked increase in the peristalsis exists as a result of disturbances of innervation. In the macroscopic and microscopic examination one has also to watch for hardened fecal masses and other concretions, foreign bodies, the ova of parasites and parasites themselves, which may give rise to acute enteritis. If the latter causes high fever, swelling of the spleen, nephritis, or pains and swelling in the muscles and joints, the diagnosis of acute infectious enteritis is logical (concerning localization of the catarrhs, see Symptomatology).

Prognosis.

This is in most cases a favorable one, especially in previously healthy adults; with proper conduct of the patients, as well as the avoidance of injurious matter, complete recovery is generally attained after a few days. In the case of small children, adults convalescing from an exhaustive sickness, and weak elderly persons the prognosis should be guarded, as in these cases it may lead to death, especially when the stomach also is affected. Improper diet and unhygienic mode of life cause new injurious substances to act repeatedly upon the inflamed mucous membrane; relapses and a transition into chronic catarrh are the consequence.

Relapses may likewise occur and chronic catarrh may be developed when the appearance of acute catarrhs is favored by a somewhat long-continued venous hyperemia of the mucous membrane of the intestines as a result of chronic diseases of the heart, lungs, and liver.

Therapeutics.

In the treatment, rest and diet are the indispensable requisites. In mild cases of enteritis a medicinal treatment is generally unnecessary, and the province of the physician is restricted to the keeping of injurious influences away from the intestines and the prescribing of an easily digestible diet, taken in small quantities, and the assimilation of which will tax the activity of the inflamed intestines as little as possible. If the state of nutrition is good, and especially when the stomach is also affected, a total abstinence from food for from twenty-four to thirty-six hours is often very effective with adults. If there is fever, the patient must rest in bed, and this is to be recommended also for cases without fever, in moderately severe and severe cases, especially for children. The diet to be prescribed consists of oatmeal porridge, barley porridge, bouillon with eggs; for drinking, boiled water with a little tea added, boiled milk in small quantities, Apollinaris, Selters waters from which the excess of CO_2 has been removed by stirring, with a little Burgundy or imported claret (Beaune) added. Very cold drinks, likewise water-ices and beer, for which patients often have a strong craving on account of their violent thirst, are to be forbidden. Eggs occasionally cause an increase in the intestinal putrefaction on account of the large amount of decomposable albumin and sulphur they contain. Likewise all those foods which are easily subject to fermentation in the stomach and intestinal canal—that is to say, fresh bread, which is to be replaced by toast, dishes made from flour, fruit, vegetables—are not to be allowed. If there is much flatulence, the use of water containing carbonic acid must be avoided. If the patient is very feeble and if the heart is weak, I allow small quantities of strong wine (Burgundy), also cognac, and, in case of collapse, mulled wine. In addition, the extremities are to be rubbed, or the patient is to be put for from twelve to fifteen minutes into a warm plunge bath. When the symptoms of acute enteritis have disappeared, a diet of easily digested meat dishes, such as boiled or broiled sweetbreads, broiled trout or blue fish, young pigeons, rice pudding, sago, and, later on, also potato purée and stale bread, is first allowed, and even after complete recovery the patient must for some days have regard for the sensitiveness of the intestines, and foods which are difficult of digestion, such as rye bread, whole potatoes, cabbage, and other vegetables, are to be avoided. If there are severe pains in the abdomen, hot poultices, frequently renewed, generally do good service, and at night Priessnitz bandages may be used for colicky pains. If the pains are very frequent and intense, I allow morphin subcutaneously or opium internally. The acute enteritis may have been caused by incorrect diet, such as a too large meal or the partaking of decayed foods. In these cases the duration of the disease may often be shortened by giving a purgative early—calomel is best, 0.3 to 0.5 for adults ; 0.02 to 0.05 for children. Tablet triturates of gr. $\frac{1}{10}$ of calomel every hour serve this purpose well. For more rapid action I advise a bottle of effervescent citrate of magnesia ; also soluble elastic capsules of pure castor oil, or castor oil in emulsion as follows :

℞. Castor oil,	30 c.c.	(1 ounce)
Gum arabic,	5 gm.	(75 grains)
Oil of peppermint,	3 drops	
Distilled water,	120 c.c.	(4 ounces).

SIG.—A tablespoonful at a time.

Purgatives are also prescribed when it is suspected that in spite of frequent liquid dejections masses of decomposed ingesta, etc., are retained in the intestines. If they are suspected in the large intestine, large colon enemata or irrigations will be of service (water, one quart, ichthyol, one dram). If in spite of complete emptying of the intestine the diarrhea continues, and if the patient still complains of pains, opium is to be prescribed in rather large doses, in the form of tincture of opium, from ten to fifteen drops in a dose, eventually several times a day. If this is vomited, I give a mixture of muriatic acid with morphin muriate, which, according to Fleischer's experiences, agrees better with children than opium. The denarcotized extract of opium, $\frac{1}{4}$ of a grain, together with ten grains of bismuth subgallate and ten grains of tannigen thrice daily has my preference for adults. In cases of persistent vomiting small amounts —one-half to two ounces—of cold effervescent champagne often act charmingly; all food should be withheld. A considerable quantity of indican in the urine, together with the emission of offensive flatus and meteorism, point to a somewhat severe fermentation and putrefaction in the intestine. Large colon irrigations with ichthyol (one dram to one quart H_2O) or a saturated solution of thymol in warm water are then very serviceable. Disinfectants given by the mouth have mostly an uncertain effect, since they are generally already absorbed in the stomach, and one does not know whether they are partially secreted again with the digestive juices into the intestine. Likewise the effect of astringents, which are very popular in catarrh, is uncertain, for the same reasons. Nitrate of silver is changed already in the stomach into insoluble silver chlorid if HCl is secreted. The astringents do good service in the local treatment of catarrhs of the lower section of the intestine when used as irrigations. In acute catarrh of the descending colon and the rectum I prescribe first a cleansing enema followed by an irrigation with one dram of ichthyol to one quart of warm water, or tannic acid (0.5 to 1 : 500 to 1000) or boric acid of the same dilution.

With severe tenesmus, suppositories of cacao-butter with opium or extract of belladonna are advised; also hip-baths. If the acute enteritis is caused by helminthæ, anthelmintic remedies are to be prescribed (see Chap. XXXIII). Constipation, which sometimes occurs in conjunction with acute catarrh, but which generally lasts only a few days, should be left to take care of itself and should by no means be treated with purgatives, which are proper only when the constipation is very stubborn and protracted. Large enemata of eight ounces of olive oil are safest for this condition. If it is caused by a spasm of the muscular system of the intestines, the use of opium is unavoidable (see Spastic Constipation). With secondary catarrh the therapy should naturally have regard for the primary disease—for instance, with malarial catarrh, quinin would be logical; with valvular cardiac diseases digitalis or strychnin would be indicated.

2. CHRONIC ENTERITIS.

Pathology.

The color of the intestine may vary from a pale red or gray to a dark or black pigmentation, which is most intense on the villi and lymph-nodules.* The mucosa is considerably swollen, and there are evidences of an extensive serous infiltration. The histological changes in many types of chronic catarrh consist of the same anatomical conditions as are found in acute catarrh. In most cases of the chronic type the small round-cell accumulations, which are so characteristic of the acute type, are lost, and an increase in the development of the connective tissue becomes more and more evident. According to Nothnagel, idiopathic and secondary chronic catarrh may exist for a long time, and yet the glands may be long and well developed, the only changes pointing to a chronic enteritis being accumulations of pigment in the glandular interstices or along the muscularis mucosæ, and a slight broadening of the connective tissue in the interstices. The enteritis may be *hyperplastic* or *atrophic*. Sometimes the chronic enteritis expresses itself in an abundant infiltration of round cells and leukocytes into the interstitial connective tissue; from this a connective tissue hyperplasia may develop. In the hypertrophy of the mucosa the gland tubules are elongated, serpentine, and expanded. The hypertrophy may extend for a considerable distance and cause constriction of the intestinal lumen. Occasionally the mouths of the glands are closed up, thus preventing an escape of the secretion, which accumulates and causes small cystic dilations (chronic cystic enteritis). In other types the extensive proliferation of the connective tissue causes the formation of polypoid growths (enteritis polyposa). The lymph-nodules may undergo hyperplastic enlargement involving the solitary as well as the agminated glands of both the large and the small intestine, either singly or together (enteritis nodularis of Orth).

The atrophic form of enteritis will be described in chapter XXIX. The glands are atrophied, the mucosa and submucosa are thinned out, the villi waste away, and the intestinal lymph-nodules are at first swollen and surrounded by a hyperemic area, but later on they rupture and give rise to follicular catarrhal ulcers. Catarrhal ulcers may also develop from desquamation and atrophy of the epithelium, which gives rise to superficial erosions which, though small in the beginning, gradually coalesce and penetrate into the deeper layers of the mucosa.

---

* Fleming has emphasized that the expression "*follicles*" for the lymph-nodules of the intestines is improper. Follicle means a sac, tube, or bag. It can therefore not be an appropriate designation for the lymph-nodules.

Symptomatology and Diagnosis of Chronic Enteritis.

There are sufferers from chronic enteritis who have no subjective complaints, but most of them experience a general diffuse soreness and distress in the abdomen. The **subjective symptoms** most frequently complained of are pain, pressure, fullness in the abdomen, and borborygmus (rumbling noises in the bowels). These sensations may be present constantly or occur spontaneously as a result of taking in food or of bowel evacuations. Abdominal pain and distress occur in paroxysms after anything debilitating the general condition—for instance, severe bodily exertion, psychic emotions, traumatism, errors in diet, or after catching cold. The sensations are attributed partly to the inflammatory process itself, to the distention of the intestine by gases, and to the absorption of toxins from the intestine. These patients may even have reflex gastric symptoms when the stomach is really not diseased; thus they may feel an annoying nausea which at times leads to vomiting. Tenesmus is an occasional symptom. So far we have referred to the symptoms of *typical cases* only. There are *atypical cases*, in which the complaints are limited to uncomfortable painful sensations distributed over the entire abdomen, or localized along the anatomical course of the colon, in the right iliac fossæ, or around the umbilicus. Sometimes these sensations rapidly disappear after the discharge of gases by the rectum. In such cases the evacuations should be carefully studied macroscopically and microscopically, and an exact clinical examination of the patient undertaken, for masked cases of chronic enteritis or of old enteritis imperfectly healed often present themselves under this guise.

The **objective signs** may, for practical purposes, be considered under two headings: Those derived from the palpation of the abdomen, and those from examination of the dejections. Palpation may reveal localized painfulness, the splashing of liquid within the colon or intestinal loops, or hard fecal masses in the constipated form. The two most important features of the stool to be examined for are the presence of abnormal constituents, particularly bile and mucus, and the amount of undigested food substances. Concerning the nature of the stool, the same description appertains as that given for acute enteritis. The reaction is faintly alkaline to cochineal, methyl-orange, and red lacmoid. When the passages are numerous, they generally react faintly alkaline to phenolphthalein at the same time. The most characteristic sign of chronic enteritis is the presence of mucus, the amount and character of which may vary with the localization of the enteritis. In chronic enteritis that has progressed to intestinal atrophy mucus is almost entirely absent from the stool.*

Blood and pus are not found in the dejections of chronic enteritis. There can be no doubt that the exactness of the clinical description of chronic enteritis has suffered considerably from the fact that it has frequently been diagnosed where it really did not exist. Chronic diarrhea, intestinal dyspepsia, various intestinal neuroses, and habitual constipation are most frequently confounded with it. In order to understand better the extreme variation in the number and character of the dejections in chronic enteritis, it is important to bear in mind that the nervous end-organs in the intestinal wall suffer considerably by the inflammatory process, and that their automatic function is severely disturbed. This interference with the automatic function may express itself in a reduced, as well as in an exaggerated, excitability to stimuli.

There is no type or form of the dejections which could be designated as characteristic of chronic enteritis. There are very bizarre and eccentric types which for a long time may frustrate the efforts of the ablest diagnostician. These are found especially in those cases where the enteritis is combined with an affection of the central or peripheral nervous system or of the special nervous apparatus of the intestine itself. Practitioners are in the habit of systematizing things which they desire to comprehend more intelligently into schemes, classes, or types. This is a particularly dangerous method when applied to enteritis; nevertheless, with a view to obtaining a more lucid representation of this subject, we will attempt a classification of the types of chronic enteritis that are most likely to present themselves to the general practitioner.

---

* Nothnagel, "Beiträge zur Physiologie u. Pathologie des Darmes," Berlin, 1884, S. 191.

Clinical Types of Chronic Enteritis.

Type 1.—In this class the patients have but one evacuation in twenty-four hours. It is mostly unformed, soft, and of the consistence of putty. The diagnosis in these cases is possible by the macroscopic and microscopic examination of the stool and the subjective and objective symptoms.

Type 2.—Here we are confronted with the most pronounced constipation—a hard and formed evacuation occurs only once in from two to four days. In some cases laxatives are necessary in order to effect a bowel movement. Mucus will be found in the stools on careful examination.

Type 3.—These cases have several markedly diarrheic evacuations in twenty-four hours, and may show this condition for months. While in type 2 the enteritis is limited to a part or the whole of the small intestine, we have an involvement of both the colon and the small intestine in type 3, for in the dejections of this latter type we find the yellow mucous granules described under acute enteritis, epithelial and round cells, stained by bile, or a diffuse reaction of bile pigment. Nothnagel explains this diarrhea by assuming an irritation which is exerted upon the inflamed mucosa of the colon by incompletely digested food and abnormal products of fermentation and putrefaction. In this third type of cases the peristalsis is permanently increased.

Type 4.—In these patients constipation and diarrhea alternate, or we may have at various times all three conditions—namely, constipation, normal evacuations, and diarrhea. It may be assumed that the physiological irritability of the intestinal nervous apparatus is well preserved in these cases, for, although the essential tendency in all cases of this type is toward constipation, intestinal putrefaction and fermentation are still capable of producing an increased peristalsis and diarrhea. In my experience the larger number of cases of chronic enteritis belong to this fourth type, which shall therefore be described in detail. The periodicity of the normal evacuations, constipation, or diarrhea may vary considerably. One or the other condition may exist for days, weeks, and months, before it changes to another condition. The irregularity and indefiniteness of the periods of diarrhea are due to accidental injurious conditions, such as we have mentioned in the etiology, and which lead to an acute aggravation of the chronic process. The order of change of these conditions of the evacuations is often as follows: For two or three consecutive days the patient has normal evacuations; on the fourth day, after premonitory abdominal pains and borborygmi, he will experience from four to eight diarrheic evacuations, which are again followed by constipation. Here we have normal evacuation followed by diarrhea, and this in turn followed by constipation. Another order is the following: For two or three days the patient has daily a very constipated passage; then follow from four to eight days of very soft evacuations with an intimate admixture of mucus, associated with pain and tenesmus. On the following day the patient has constipation, followed by two or three days of very hard stool. Here we have constipation, diarrhea, and constipation. These various types may repeat with surprising regularity. The alternation of the conditions of the dejection, as we have described in this type 4, is more characteristic of chronic enteritis than of any other condition of the stool with which we are familiar.

Test Lavage of the Colon.—This is executed by inserting a Langdon colon tube about 80 cm. long into the colon. A large glass funnel of the capacity of one liter is attached to the end of the tube. Immediately after the latter is inserted into the rectum the funnel is gradually raised so that a stream of water continually issues from the end of the tube as it is slowly pushed forward. The water pouring out from the end of the tube pushes away folds of mucous membrane and excremental matter that might obstruct its progress. In this manner the tube may be passed through the sigmoid flexure and transverse colon up to the cecum. In case difficulty with the Langdon tube is experienced, a tube containing a very pliable metallic spiral has been successfully employed by the author. It was presented to him by Dr. Franz Kuhn, of Giessen, and has been described by him in the "Archiv f. Verdauungs-Krankheiten," Bd. III, S. 19. This tube can be recognized within the colon when the patient is placed before the X-ray apparatus, by means of the fluoroscope, and I have been able to demonstrate in this manner that it can readily be introduced into the cecum and that it is possible to wash out the cecum in this manner.

When the examination of feces does not give satisfactory results, experimental lavage of the colon has often, in my experience, given further information, not only concerning the contents, but also concerning the condition of the mucosa of the colon.

Under normal conditions the experimental lavage of the colon shows very slight admixture of the water with fecal particles, mucus, and epithelial detritus, but the water very often returns almost clear. In the various forms of colitis, however, mucus is abundant in the lavage water, and it also contains fragments of the colon mucosa which are available for microscopic examination. On several occasions I have succeeded in finding fragments of neoplasms situated in the transverse colon in this manner. The method is available for the diagnosis of all the various types of colitis.

Enteritis and Diarrhea in Connection with Achylia Gastrica.—In the theoretical portion of this work I have abstracted the experiments of Pawloff ("Die Arbeit der Verdauungs-Drüsen," S. 165), in which it was conclusively shown that the HCl of the gastric secretion is the specific excitant to the secretion of the pancreas. It is therefore plausible that the diarrhea and the consequent enteritis in the sequence of achylia gastrica may be brought about in two ways: (1) By the total absence of gastric digestion and solution of the proteids, together with the inhibition of the pancreatic secretion, owing to the absence of its physiological stimulus (HCl), and (2) by an extension of the gastritis or gastric atrophy to the mucosa of the intestinal canal. When the intestinal symptoms appear in the course of this complication, the gastric symptoms may become completely submerged, the chronic diarrheas being the main distress. In the dejections the substances taken in as food are largely found undigested. Meat, connective tissue, carbohydrate foods, and sometimes even the fats are found unaltered. The secretory function of the stomach is, as a rule, completely lost, and HCl and ferments are absent. The motor function, however, is well preserved. Biedert, who suffers from achylia gastrica himself, has given an interesting autographic description of his personal history of these intestinal complications (Biedert and Langermann, "Diätetik u. Kochbuch," 1895). In the following I will briefly describe a typical case of this kind:

Mrs. G. C. G., aged fifty, has five children; has been treated by me in 1888 for neurasthenia gastrica. At that time the chemical analysis gave the following results: Free HCl 20, combined HCl 12, total acidity 38. In 1895 she returned with symptoms of chronic gastritis. Much mucus was found in the stomach every morning at lavage before breakfast. She had lost twenty pounds in two months at that period. Appetite was variable, but generally poor. Bowels were regular. During the first attack, when she was treated for neurasthenia gastrica, she suffered from constipation. Since 1895 the gastric symptoms have become more and more aggravated, the amount of free HCl less and less, and now there is evidence of a progressive chronic gastritis. On June 10, 1897, free and combined HCl were both absent, but the bowels were still regular. I did not see the patient again until October 9, 1899, when she consulted me again for severe diarrhea, which she informed me had existed at intervals for two years. She had four passages during the day, and two or three movements of the bowels during the night. These generally occurred between three and five o'clock. The evacuations showed much blood in the passages, but no mucus. Chemical analysis of the gastric contents showed evidences of complete achylia. After a meal consisting of a Hamburg beefsteak, mashed potatoes, and rice, the evacuation on the following morning showed an abundance of unaltered beef muscle-fibers and also unaltered particles of potato and rice. The connective tissue was very easily recognizable among the undigested meat-fibers. Milk was not curdled by the gastric filtrate. A curious observation was made on this patient during the treatment. It was found that her stomach could not tolerate the smallest dose of dilute HCl. As I had seen very favorable results in these cases from the therapeutic use of dilute HCl,—not from its digestive effect, for which enormous quantities would be necessary, but perhaps for the stimulating influence it exerts upon the secretion of the pancreas,—I persisted in its use, beginning with one drop of dilute HCl three times a day. In four months she gradually accustomed herself to taking sixty drops three times a day, with the result that the undigested meat particles in the evacuation have become less and less, the diarrhea has been checked, and the stools are rather constipated. This, of course, could not be attributed to any direct digestive influence of the HCl, but is undoubtedly attributable to a better secretion of pancreatic juice which has been brought about by the treatment. When the diarrhea was severe, she received, in addition to the foregoing treatment, fifteen grains of bismuth subgallate and $\frac{1}{30}$ of a grain of strychnin sulphate three times a day. The diet was rigidly controlled according to principles explained in another chapter.

For differential diagnosis see section on Neurasthenia Intestinalis and Intestinal Dystrypsia.

Prognosis.

The prognosis of chronic enteritis is dependent upon the cause, duration, intensity, and extent of the disease. It is a grave disease when it occurs as a complication of other abnormalities. The prognosis is serious in old age and early childhood. A chronic enteritis which has involved the entire ileum and colon and has existed longer than three years is, in my experience, never entirely curable. The anatomical changes in the mucosa that occur in this time render the possibility of a restitution to integrity inconceivable. Even a very intense chronic catarrh, limited to the small intestine or to the colon only, that has existed longer than two years is, in my opinion, very rarely entirely cured, because in such cases a disposition to relapses is, as a rule, observed; but there can be no doubt that more limited catarrhal inflammations of the intestine can be cured entirely, especially if they are taken in time. Chronic enteritis occurring in aged patients beyond the sixtieth year are never healed perfectly, but continue with remissions and exacerbations through life. In many cases where a restitution to integrity is impossible, treatment should not be given up because it is frequently possible to restore the functional power of the intestine and prevent loss of weight by proper treatment, even in these cases. In extensive atrophy of the intestinal mucosa the prognosis as regards life is unfavorable.

Treatment of Chronic Enteritis.

It is of practical value to decide whether we are confronted by: (1) A chronic enteritis of the small or the large intestine, or both; (2) a chronic enteritis of comparatively short duration—say existing for a year or less—where there is still hope for complete cure; (3) where the disease has existed for more than two years. Colitis can be very effectively treated locally, and medications by the mouth should, whenever possible, be excluded. Local treatment, rest, and carefully selected diet are, in my experience, very successful factors in the treatment of colitis. But enteritis involving the small intestine can not be reached by irrigations from the colon, and a limited amount of medication by the mouth is unavoidable. The treatment of chronic enteritis of comparatively short duration must necessarily differ from that of enteritis of long duration, because in the former the general body nutrition is still well preserved, and the practitioner will feel justified then to impose a diet more restricted than when the disease has lasted for a long time and metabolism has suffered seriously from its ravages.

The most far-reaching principle in the treatment of chronic enteritis in general is *cleanliness of the intestines, as much as is consistent with adequate nourishment.* The value of the therapeutic methods may be stated in the following order: Rest, intestinal cleansing, diet, external application of warm fomentations, hydropathic treatment, and certain medicines.

Rest is best secured by enforcing the reclining position in bed, which at the same time will avoid dietetic errors and aggravations by cold.

Intestinal cleansing is carried out by high colon irrigations, for which plain warm water is best suited. If the enteritis is limited to the colon, the following substances may be added to the water: Nitrate of silver, 0.2 gm. to 1 liter (1 quart); salicylic acid, 1 gm. to 1 liter; tannin, 2 gm. to 1 liter; boric acid, 5 gm. to 1 liter; tannigen, 3 gm. to 1 liter; the tannigen must be rendered soluble by an equal quantity of sodium bicarbonate. In my practice ichthyol, 4 gm. to 1 liter, has proved very helpful in the irrigation treatment of colitis. When it becomes necessary to cleanse the intestine from above by medicine, I use only two remedies: castor oil and calomel. The calomel is best given in doses of ½ of a grain every two hours, until the purgative effect is brought about, but I really prefer castor oil to calomel—it should be given in doses of one teaspoonful when there is diarrhea, and one tablespoonful when there is constipation. Unfortunately, quite a number of patients feel so irrepressible a disgust for this really very useful medicine that it is difficult for them to take it. In that case it is sometimes possible to give it after it has been prepared by the druggist with compound syrup of sarsaparilla or in capsules of the pure soluble gelatin.

The **dietetics** of enteritis must aim to accomplish the following results: (1) To avoid such food as might possibly increase the disease, either by already containing toxins or bacteria before it is eaten, or by the possibility of its decomposing and forming irritating substances after it is ingested. (2) The selection of very digestible and assimilable food. (3) Selection of food which either in itself or by the changes it undergoes in the digestive canal shall have a constipating, a laxative, or antiseptic effect, as may be indicated.

Food and drink should never be taken very hot or very cold. It is not always desirable to select such food as can exert an astringent effect or check secretion, for in the majority of forms of enteritis the fundamental condition is constipation and not diarrhea. Whenever it is possible to effect movement of the bowels by a dietetic substance, this should be preferred to a medicinal purgative. Sometimes very bizarre and grotesque combinations have been known to exert this influence. I have known one case in which very finely cut stewed string-beans regularly produced an evacuation; in another it was buttermilk; in a third, stewed apples. In his "Diagnostik und Therapie der Darm-Krankheiten," page 232, Boas speaks only of the use of foods having an astringent or constipating effect. There are, however, in my opinion, many types of chronic enteritis in which laxative food is desirable. There are only three types of enteritis which are associated with diarrhea; these are: (1) The acute enterocolitis; (2) the acute colitis; (3) the chronic enterocolitis involving the entire small intestine and the entire colon. But a majority of the chronic forms as they come under observation are associated with constipation. Thus, chronic colitis is accompanied by constipation. Chronic enteritis involving the small intestine only is associated with constipation, and even when, in addition to this, the cecum and ascending colon are involved in the chronic catarrh, the prevailing condition will be that of constipation. In those forms of enteritis in which diarrhea and constipation alternate there is really more danger in the constipation than in the state of diarrhea, for the retention of hard feces constitutes a decided stimulus to the inflamed intestine, which is in a condition of increased irritability. A guiding principle, therefore, in the dietetic treatment of chronic enteritis is never to allow the condition of constipation to persist without gentle interference. This is best done by diet.

For the dietetic and medicinal management of persistent constipation I refer to the chapter devoted to this subject, with the restriction, however, that many things can be given in chronic constipation when there is no inflammatory condition of the bowel, which must be avoided in the constipation due to enteritis. As it is, however, a great gain to be able to manage this latter type of constipation by diet, I will mention a few things that have been helpful to me for this purpose. Among the first of these is kephyr and also kumiss. These are fermentation products of milk. The preparation of kumiss is a specialty in the Russian steppes, where it is prepared from mare's milk. The kumiss ferment and the kephyr ferment effect analogous changes in milk. They effect a triple fermentation: first, a lactic acid fermentation; second, an alcohol carbonic acid fermentation; third, a partial transformation of the casein into acid albumin, hemialbumose, and peptone. On account of the carbon dioxid it contains, the kephyr is slightly effervescent, and for many patients more palatable than milk. Kephyr should never be permitted to stand longer than three days before it is used. I have obtained best results with that which was two days old, and, when used with caution, avoiding other foods that might cause distress, I have not seen any unfavorable results follow its dietetic employment in the constipation of enteritis. Kumiss and buttermilk may be used in the same way, although the latter is more likely to cause intestinal irritation.

Somatose is a yellowish powder consisting of a mixture of albumoses, soluble in water, and prepared from meat by F. Bayer, Elberfeld, Germany. In doses exceeding one dram this artificial food has a laxative effect. I have often employed it for this purpose only in constipation associated with enteritis, and as it is highly nutritious at the same time, and can not possibly cause intestinal fermentations, it can safely be recommended. Other foods useful for effecting regular movements are mentioned in chapter xv.

Dietetic Management of Diarrhea in Enteritis.—This is essentially the same as that stated in the chapter on Diarrhea. The articles of diet which exert a constipating effect owe this in general to a certain amount of tannic acid which is contained in them. The chief among these is acorn chocolate (Eichel Kakao), which is prepared according to the formula of Professor Liebreich, by Stollwerk, of Cologne. It contains nearly 2 per cent. of tannic acid, and it is really a mixture of ground acorns with the purest chocolate. Winternitz recommends decoctions of dried huckleberries (250 gm. of dried huckleberries are mixed with one liter of water; then boiled down to 750 c.c.—*i. e.*, two-thirds of the original bulk; the berries then pressed out, strained through a cloth, and the juice thus obtained added to the 750 c.c.). This can be sterilized by boiling and preserved in bottles in a cool place.

A curious dietetic effect has been noticed with regard to kephyr, and that is, in some it will cause a laxative effect and in others a constipating effect. This can be largely explained by the age of the kephyr. Kephyr which is only two days old is that which has a laxative effect, while that which is four days old has a constipating effect; it contains 0.63 per cent. of lactic acid, and, according to Wegele, remarkable cures of chronic diarrhea have been effected by it. It should be taken in doses of eight ounces at a time. About two quarts can be taken in twenty-four hours.

It is not possible to state *a priori* in each case what kind of an effect kephyr will exert upon the patient. The future use of the substance will depend upon a trial in each individual case.

The Dietetic Use of Milk in Chronic Enteritis.—Boas (*l. c.*, pp. 157 and 232) is inclined to forbid the use of milk entirely. There can be no doubt that milk should not be ordered indiscriminately. Some patients have an aversion to it and will not take it at all; in others it predisposes to diarrhea; in still others it actually provokes diarrhea. I feel assured, however, that the class of patients in whom milk disagrees are decidedly in the minority. If it is known by experience to exert a constipating effect, sterilized milk might be used to check the diarrhea, and vice versâ. It must be emphasized that, according to Soxhlet, not every kind of milk can be sterilized. If the milk has been handled very uncleanly or if a long time has elapsed between the milking and the time at which the milk reaches the kitchen, toxic changes have taken place in the milk which can not be removed by heat; or very resistant types of bacteria may have developed which can not be destroyed by heat. Before blaming the milk, therefore, it is absolutely necessary to be assured that it is sterile. Some very persistent forms of enteritis have been cured by an exclusive milk diet. In some instances where patients have a decided aversion to milk they can be persuaded to accustom themselves to it. An attempt should be made by adding such substances to the milk as may make it more palatable to the individual—a little lime-water, coffee, or tea, or a little brandy or rum, as the case may be. There can be no doubt that there are rare cases of idiosyncrasies in which milk is not well digested. For practical purposes these exceptions may be disregarded.

General Dietetic Rules for Chronic Enteritis.—The diet should, as far as possible, be semisolid or liquid; not much of fatty foods or foods prepared with fats. The food should be thoroughly chewed and insalivated. Not much should be eaten at once, but small meals should be taken about every three hours.

Menus.—Soups made of thoroughly cooked rice, sago, farina, barley, oatmeal, mondamin, stale bread, Zwieback boiled in thin bouillon, with one egg stirred into each pint of soup. Meat: not more than 125 gm. at a time of lean and tender beef or ham without gravies. Later on, boiled mutton, calf's-brain, blue fish, trout, flounder, and pike. Eggs, as a general thing, are allowable; they contain large quantities of lecithin, from which the toxalbumin cholin is liberated in case the progress of the food is much impeded through the intestine. If there are symptoms of autointoxication, therefore, the yolks of eggs should be avoided. The white of egg can still be continued. Of fat, only a limited amount of butter on bread or in soups is allowable.

Farinaceous articles: Stale bread, toast, Zwieback, thin gruel of rice, sago, farina, mondamin. Vegetables: Spinach, purée of potatoes, or peas; sometimes fresh tender cauliflower. Fruits: Only strained huckleberries. Drinks: Weak tea with very little sugar; acorn coffee and acorn chocolate; milk, diluted with lime-water if necessary, or with brandy or whisky added; water and claret or Burgundy mixed.

Foods that Must be Avoided.—All fats, with the exception of a little butter, fatty soups, bouillon, and gravy. Fat meat, pastry, sausages, and fish. All meat that is hard, boiled out, smoked, salted, or preserved. Fried eggs; eggs with bacon. All foods prepared from flour with fat and eggs. Pancakes, dumplings, black bread, fresh bread. All fruits and most vegetables. Cheese, wine, lemonade, alcoholic liquors, except by special direction. Oysters, clams, crabs, and lobsters.

DETAILED DIET-LIST FOR THE DAY.

8 A. M.—One-half pint (250 c.c.) of acorn chocolate or the same quantity of hot, rich, sterile milk; one piece toast; 20 gm. butter.

10 A. M.—One cup of soup made of bouillon, farina, and egg, very slightly salted; in addition, a small sweetbread or a cup of gelatin.

1 P. M.—One cup glutinous soup, made of rice, egg, and bouillon, to which, in case there is constipation, somatose may be added; in case of diarrhea, nutrose, eucasin, tropon, or sanatogen may be added; 125 gm. of finely minced tender beef, or one of the forms of fish allowed above—trout, flounder, blue fish, etc.; 75 gm. purée of potatoes or peas; compote of huckleberries.

4 P. M.—One cup of acorn chocolate if there is diarrhea, or tea and milk if there is constipation; one Zwieback or toast with butter.

7 P. M.—Strained soup of oatmeal, 250 gm., or kephyr two or four days old, as may be indicated.

9 P. M.—180 gm. of warm milk, kephyr, or farina soup, with Zwieback; or one glass of huckleberry lemonade, made by stirring one or two teaspoonfuls of huckleberry jelly in 250 c.c. of water, to which about one ounce of imported French Burgundy has been added.

I am aware that in some cases it will not be possible to give but a fraction of the menu detailed in the preceding, and that in others an increased amount of food can be given with impunity. The practitioner must in all cases be guided by the reaction of the intestine to the nourishment that is introduced. The nutrition of patients affected with diarrhea must be regulated by daily microscopic and macroscopic examination of the stools, and in extreme cases by such testing of the function of the intestine as has been described by Dr. Harry Adler in the chapter on Examination of the Feces. The stool-sieve of Boas greatly facilitates the examinations.

Hot fomentations are very serviceable in the treatment of this disease. They can be employed in either the dry form—for instance, by bags filled with hot salt or hot sand or the hot-water rubber bottle—or in the moist form in the form of large poultices of flaxseed or bread, or simply large pieces of gauze folded together and wrung out in hot water. Patients of the rheumatic type should, in fact, always wear a woolen abdominal bandage and woolen underwear.

Hydrotherapy.—*1. Baths.*—Personally I have observed no benefit to patients afflicted with chronic enteritis from methodical cold-bath treatment, though I have studied the effects of this treatment in a sanatorium in Germany. In chronic exudative processes of the intestine, such as, for instance, the perityphlitic exudations following appendicitis, especially such as have gotten well without operation, I have observed unmistakable improvement from methodical hot bathing. For this purpose I would, however, warmly recommend our American bathing establishments at Hot Springs, Bath County, Va., or Colorado, at Saratoga, N. Y., and Mt. Clemens, Mich. At Hot Springs, Va., and Saratoga the bathing can be combined with drinking cures at the natural springs, which abound at these hygienic resorts. In Germany the best known bathing resorts for these purposes are Reichenhall, Kissingen, Wiesbaden, and, if a combination of drinking mineral water cures and mud-baths is indicated, Carlsbad, Marienbad, and Franzensbad would be proper resorts to consider. All such bath treatment must be taken under the guidance of competent local physicians who have experience with the action of the water and the diet to be selected for each cure. For patients living near the seashore, the bath in heated sea-water will effectively replace the bathing in water at the mineral springs.

2. *Drinking of Mineral Spring Water.*—The composition, effects, and mode of drinking the various mineral spring waters have been considered in the chapter devoted to this subject, and in an exhaustive article in the author's book on "Diseases of the Stomach." A number of German authors are of the opinion that certain mineral spring waters do not only influence the symptoms favorably, but actually exert a curative influence on the anatomical process of chronic enteritis. In my opinion there are many influences in addition to that of the mineral water which affect the condition of the patient; most important among these is the fact that many patients will adhere to their diet conscientiously only when they are under treatment at one of the mineral springs. Of our native springs I favor the mild bitter water of the magnesia spring at Bedford Springs, Pa., and, above all, those of the Saratoga Springs, which contain calcium and magnesium carbonate in addition to sodium chlorid. These are the Hathorn, Congress, and Kissingen Spring at Saratoga, N. Y. There is a spring at Hot Springs, Va., which furnishes warm magnesia water to which I have become very partial, owing to a very evident curative effect this water has exerted on a number of patients suffering with chronic enteritis who drank it in connection with the baths. In my opinion the most of the foreign waters are too severely purgative to be taken for any length of time by patients afflicted with chronic enteritis whose general constitution and power of resistance have, as a rule, already been considerably reduced.

The Carlsbad Sprudel Spring should be taken in very small quantities—certainly not exceeding 50 c.c. three or four times a day. Another mineral water which has achieved a reputation in the treatment of chronic enteritis is that of Vichy in France. If more attention were paid to the scientific adaptation of diet to the treatment of enteritis at Saratoga, N. Y., I feel confident that the effects of the springs mentioned above would be greatly enhanced and that they would become dangerous rivals to the springs of Carlsbad and Vichy. The waters themselves being milder are less liable to injure the mucosa and can not exert any drastic effect.

Medicines.—These may be considered under three headings: (1) Those that inhibit or stimulate peristalsis; (2) those that have an astringent effect; (3) those that have an antiseptic and antifermentative effect. There are a number of drugs which combine the latter two,—that is, that have both an antiseptic and an astringent effect,—and in cases may even act as bland, unirritating covering media for intestinal ulcers, etc.

1. There can be no doubt that it would be better for nine-tenths of cases of enteritis if they were treated by the methods previously considered, to the total exclusion of drugs, and in defining the use of those drugs which inhibit peristalsis I consider it my duty to begin with an emphatic protest against the abuse of opium. There is still entirely too much of this drug prescribed in this country in all cases in which there is diarrhea or even the slightest abdominal pain. In general practice this drug is frequently prescribed at the onset of the treatment, when its use is justifiable only after all other means of treatment have been exhausted. In a chronic disease like this there is certainly danger of habituation to this drug. Personally, I prescribe it only after trial of the other medicines to be spoken of later on, and when there is unmistakable evidence of a spasmodic state of portions of the intestine, such as might be assumed as a consequence of irritation by hard fecal masses; but in these cases I always combine it with a laxative such as castor oil or small doses of calomel. Diarrhea and pain are, in the majority of instances, caused by decomposing and stagnating fecal masses which call for rapid evacuation and directly contraindicate the use of opium. Hot application, rest, or hot poultices, together with a high colon irrigation with one or two quarts of warm water, in my

experience rarely fail to give relief in these cases. When opium had already been prescribed by others in hospital or consultation practice, I have very frequently made the observation that the pains and diarrhea of chronic enteritis disappeared for twelve hours or perhaps for one day, and thereafter they returned again intensified. I have never observed any good results following the use of belladonna in this disease, but I have frequently met with annoying complaints of a toxic influence, together with great dryness in the throat and vertigo resulting from this drug. I prefer in all cases to employ the denarcotized extract of opium, with which I have had extensive experience in such cases as presented the indications above stated. Codein is a substitute for opium which relieves the pains, but usually does not exert so constipating an effect. There are, however, cases in which it acts as constipating as opium. The drugs which stimulate peristalsis have been described in chapter XIII; it is preferable to accomplish this by the diet whenever possible.

2. The astringents may be separated into (*a*) vegetable and (*b*) mineral or metallic astringents. The vegetable astringents owe their effect to the presence of tannin. Examples of these are catechu, kino, cascarilla, rhatany, calumba—all of which can be dispensed with and effectively replaced by tannin itself, which, however, should never be given for more than three or four days at a time, and not exceeding half a grain at a dose. It must be emphasized that all remedies containing tannic acid have a deleterious effect on the stomach. Among the mineral and metallic astringents most frequently used are alum, acetate of lead, sulphate of zinc, nitrate of silver, and liquor of the sesquichlorid of iron. I would caution against the use of all these except, perhaps, of nitrate of silver, which occasionally produces a good effect. It is best used in the form of irrigations of the colon, as already described, but in rare instances its use is followed by improvement if given in the form of a hard pill—¼ of a grain three times a day. I have had four cases of membranous colitis in which this condition was distinctly aggravated by irrigations with weak solutions of nitrate of silver, and I would caution against its use in this disease.

3. Antiseptic and antifermentative drugs: This class comprises a very large number of drugs concerning the therapeutic value of which

experience justifies us to occupy the standpoint of conservatism. Among the most useful are calomel, bismuth salicylate, and betanaphthol bismuth. Magnesium salicylate, in doses of 0.2 gm., has been recommended by Huchard and Mathieu for the enteritis connected with constipation. I have had personal experience with these and benzonaphthol, in doses of 0.5 gm. thrice daily. Also with resorcin resublimate—5 per cent. solution in a mixture of water and brandy; dose, one tablespoonful from three to six times a day. Tannin most likely owes its effect to its antiseptic qualities. Recently a number of tannin combinations have been brought on the market which apparently possess real therapeutic virtues. The most useful of these, in my experience, has been tannigen (acetyl of tannin, dose, 0.2 to 0.5 gm. thrice daily). Other compounds of this character are tannopin and tannalbin. With the last two I have had a limited personal experience. Debove and Rosenheim recommend silicate of magnesium—150 gm. dissolved in milk or aromatic infusion given in the twenty-four hours. Boas and Jaworski ("Therapeut. Monatsh.," 1898, H. 2) speak very highly of the carbonate and phosphate of calcium. Boas recommends the following:

℞.	Calcium carbonate,	25 gm.	(6½ drams)
	Calcium phosphate,	25 "	(6½ drams)
	Betanaphthol bismuth,	5 "	(75 grains).

DOSE.—One teaspoonful thrice daily.

This combination is at least harmless, and often sufficed to check putrefactive diarrhea in my cases. Jaworski recommends the following artificial carbonated calcium water:

℞.	Calcium carbonate,	2 gm.	(30 grains)
	Calcium salicylate,	2 "	(30 grains)
	Carbonated water,	1 liter	(1 quart).

This is the milder effervescent calcium solution. He also recommends a stronger, containing four grams of each of the salts to one liter of carbonated water. Of these solutions four ounces of the stronger are taken on an empty stomach in the morning, and four ounces of the weaker three times daily after meals. These formulas of Boas and Jaworski are very helpful in such cases in which the enteritis is associated with gastric hyperacidity. When there is achylia gastrica, I approve of the use of dilute HCl in doses of from forty to sixty drops after meals. The following formulas are recommended for special symptoms:

For flatulence and intestinal autointoxication:

℞.	Benzonaphthol,		
	Resorcin,		
	Bismuth salicylate, of each,	5 gm.	(75 grains)
	Phosphate of calcium,	10 "	(150 grains).
M.	DIRECTIONS.—One-half teaspoonful after meals three times a day.		

For pain with profuse liquid stools:

℞.	Denarcotized extract of opium,	0.2 gm.	(3 grains)
	Tannigen,	2.6 "	(2 scruples)
	Bismuth subgallate,	4.0 "	(1 dram).
M.	DIRECTIONS.—Make twelve capsules. One capsule thrice daily.		

For gastric disturbances associated with enteritis and loss of appetite (HCl must be supplied if it is shown to be absent in the test-meal):

℞.	Strychnin sulphate,	0.02 gm.	(⅓ grain)
	Fluid extract of condurango,	64.00 c.c.	(2 ounces)
	Essence of calisaya (P. D. & Co.),	48.00 c.c.	(1½ ounces)
	Elixir of gentian, enough to make	180.00 c.c.	(6 ounces).
M.	DIRECTIONS.—One tablespoonful thrice daily.		

In concluding the medicinal treatment, I wish to emphasize that the practical value of the treatment by astringents is a very doubtful one, and that bismuth subnitrate and bismuth subgallate can be effectively administered by rectal enemata—one dram of the powder is injected suspended in half a pint of warm water. Bismuth subnitrate has been found by me and my assistants in the stomach of patients, and has been partially regained from that organ by gastric lavage after it had been injected into the rectum six to ten hours previously. For persistent obstruction of the colon by hard fecal masses the large oil enemata, as first recommended by Fleiner, can not be surpassed in efficacy by any other local medication—eight ounces of cotton-seed oil or olive oil are injected high up into the colon by the Langdon tube and retained as long as possible.

3. PHLEGMONOUS ENTERITIS (PURULENT INFLAMMATION OF THE INTESTINES).

This very rare disease is believed to be a suppurative inflammation involving principally the submucosa and muscularis of the intestine. Its extreme rarity may be judged from the fact that

J. J. Woodward ("The Medical and Surgical History of the War of the Rebellion," vol. 1, "Medical History") does not report a single case of phlegmonous enteritis among 1,739,135 acute and chronic cases of intestinal diseases, among which occurred 44,558 deaths from May 1, 1861, to June 30, 1866. In Section III of this phenomenal work Woodward has compiled the morbid appearances observed in 878 autopsies. It is not likely that the presence of so marked a histological condition as phlegmonous enteritis would have escaped this acute observer. It is true that Cullen (Wm. Cullen, "Nozologia methodica," third edition, by John Thompson, Edinburgh, 1820) has subdivided idiopathic enteritis into the phlegmonous and erythematous ("phlegmonodœa" and "erythematica"), but in his chapter on Enteritis in his "First Lines of Practice of Physic" he treats only of the phlegmonous variety.

It is very evident, on studying these writings, that although he used the term, Cullen really had no knowledge of phlegmonous enteritis as we conceive it at the present day. The description he gives under this name is applicable to peritonitis alone.

Phlegmonous enteritis is a secondary phenomenon in consequence of other intestinal diseases, particularly carcinomata, ulcers of tubercular, dysenteric, and embolic origin, and occasionally as a consequence of strangulated herniæ and intussusceptions; in all these conditions the diagnosis will probably not be made even if a secondary phlegmonous enteritis exists.

The **etiology** of this disease is unknown, but it is probably of an infectious origin. The case that has been most quoted in this connection was reported by Bellfrage and Hedenius (Virchow and Hirsch's "Jahresbericht," 1876). The patient was a man fifty-two years old, who was suddenly taken with symptoms of peritonitis. A crural hernia, apparently not replaceable, was discovered, but when herniotomy was undertaken, this proved to be a fatty tumor. Death occurred on the third day. An intense phlegmonous inflammation had invaded the wall of the jejunum for a distance of eighteen centimeters. The mucosa was of a grayish-yellow color, and presented a large number of bright yellow spots which in various places showed superficial ulceration. The base of the ulcers presented a loose connective tissue infiltrated with pus. There was marked thickening of the submucosa and muscularis, which in some places had swollen to eight centimeters (3¼ inches) in thickness. There was a diffuse acute peritonitis.

The **clinical symptomatology** can not be given for lack of a sufficient number of observations. The disease will probably escape recognition, and thus I can dispense with the consideration of the diagnosis. The **treatment,** therefore, need not be considered in detail. It is not probable that the general practitioner will meet with this disease in private practice—at Nothnagel's clinic no such case has ever been observed. I have not seen such a condition personally. The treatment, if any, would probably coincide with that of an intense acute enteritis.

4. DIPHTHERIC, CROUPOUS, OR PSEUDOMEMBRANOUS ENTERITIS—DIPHTHERIC DYSENTERY OR ULCERATIVE COLITIS.

These designations are given to the diseases of the intestine described in this chapter simply because they are characterized by the formation of a pseudomembrane. According to our modern conceptions we should, strictly speaking, designate only such intestinal diseases as diphtheric which are caused by the Löffler bacillus. The terms diphtheric and croupous, or pseudomembranous, enteritis find their justification, therefore, not in the etiology, but in the gross anatomical appearance—the formation of a false membrane involving the mucosa and deeper layers of the intestine.

It is highly probable that pseudomembranous enteritis is also caused by bacteria, which, however, up to the present, have not yet been isolated in pure culture.

The human intestine is in very rare instances the seat of *genuine diphtheria* caused by the *Löffler bacillus*. The pathological histology has been described by Ssmirnow ("Virchow's Archiv," Bd. CXIII), who, in accordance with the views of his teacher, von Recklinghausen, has described two groups of genuine intestinal diphtheria. In the first group there is an exudation of fibrinous material, with loosening and detachment of the epithelium; in the second group of undoubted diphtheric enteritis the characteristic inflammatory phenomena are wanting, but extensive hyaline degeneration of the mucous and submucous tissue, with swelling and hyaline transformation of the epithelium into a fibrinous network ending in necrosis of the connective tissue and mucosa, are present. Clinically this condition is difficult of recognition. The symptoms, of course, are the same as in simple acute dysentery. According to Baginsky (Nothnagel's "Specielle Pathologie u. Therapie," Bd. II, "Diphtherie," S. 99), the principal seat of genuine intestinal diphtheria is in the colon and above the rectum, where it appears in the form of stripes on a hemorrhagic background of the congested intestinal mucosa. It is therefore advisable, where diphtheria is known to exist in the throat, to examine the rectum by inspection, if possible, in cases with symptoms of enteritis. The intestinal diphtheria is always a secondary form, and the treatment will coincide with that of the primary affection, and therefore depends upon the success of the injections of antidiphtheric serum.

This brief reference will be sufficient for the rare form of genuine diphtheria of the intestine, and we shall now resume consideration of the main subject of this chapter.

Classification.

The clinical and pathological features of diphtheric inflammation of the colon will be more accurately described in chapter XX on Dysentery. The diseases which may exhibit this anatomical picture at the autopsy may be primary or secondary. The *primary type* is characterized by hyperplasia and infiltration of the walls of the colon, leading to ulceration and sloughing of the mucosa, with more or less intractable diarrhea. In the milder cases the surface of the folds of the mucosa and of the colon are covered with a thin yellow exudate. In the severer cases there is an extensive necrosis—the entire mucosa of the colon may gradually be transformed into a yellowish layer in which the characteristic histological elements of the mucosa are no longer recognizable. The necrosis may be superficial, involving only the glandular layer of the mucosa, or it may be deep, penetrating to, and even perforating, the peritoneum. The *secondary form* occurs as a terminal colitis in the sequence of many acute and chronic diseases which we shall enumerate presently.

Diphtheric Colitis Due to Mercury.

It has long been known that calomel and blue mass given for laxative purposes were capable of exerting, even in small doses, very deleterious effects upon the intestinal mucosa in susceptible individuals.

From a careful study of Woodward's "Medical and Surgical History of the War of the Rebellion," and abundant experience in the treatment of various types of dysentery and colitis occurring in our southern States, and from the experience gained in two epidemics of these diseases at Bay View Asylum, Baltimore, during 1884 to 1888, when I was physician-in-charge of this institution, I have concluded that mercury and all its preparations, with the exception of calomel, can not exert any favorable influence on the morbid processes involved in these diseases, but, on the contrary, frequently aggravates them, and that, therefore, these drugs should be excluded from the treatment. Even calomel exerts its favorable effect by its purgative influence, and has maintained its place in the therapeutics of this disease mainly because of its ready administration.

It has long been known that severe and intense intestinal symptoms may occur after inunction with mercurial ointment, after subcutaneous injection of mercurial preparation, and after administration of calomel. I have been consulted in two cases of dysentery with frequent bloody stools and high fever which had followed a uterine irrigation with a solution of corrosive sublimate 1 : 5000. These attacks ceased entirely when the irrigations were suspended and the patient placed on a milk diet. The antiseptic treatment of wounds and the preparations for operation which have necessitated the use of corrosive sublimate have frequently been associated with acute intestinal symptoms. Jacusiel ("Berlin. klin. Wochenschr.," 1887, S. 253) has observed very severe intestinal disturbances after he had personally executed an inunction with two grams of mercurial ointment. From this it is evident that in certain predisposed cases it is not necessary that especially large quantities of mercury be incorporated in order to exert a harmful influence. No doubt certain preexisting conditions may favor the deleterious influence of the mercury—such as, for instance, already existing enteritis, general debility, and anemia.

Two theories have been offered in explanation of this effect of mercury. The first one, that advanced by Overbeck, suggests that the metal is secreted through the liver and reaches the intestine in the bile. The second theory holds that the effect is exerted by the blood. According to Rossbach, however, mercury circulating in the blood is not considered capable of producing inflammation, and is claimed to occur there in the form of an oxyalbuminate of mercury.

Constipation as a Causative Factor of Pseudomembranous Enteritis.

There is a form of pseudomembranous dysentery which develops above intestinal stenoses which has been assigned partly to the effect of the stenosis itself and partly to the effect of the stagnating masses above it. This leads me to refer to the factor of constipation in the production of this disease. Virchow ("Virchow's Archiv," Bd. II u. V) held that dysentery could develop through the effect of substances which are contained in the intestine, exerting an irritating influence upon the mucosa, which is already in a state of catarrhal affection. In other words, he asserted that a simple enteritis or catarrhal dysentery could be converted into diphtheric dysentery by the presence of putrefactive material in the intestine. It is conceivable that putrefying and stagnating intestinal contents may exert this effect by chemical substances, toxins, or by the bacteria contained in them. Annesley and Trousseau also attach importance to the presence of stagnating fecal masses in the causation of ulcerative colitis. Nothnagel does not commit himself on this point. The great frequency of persistent coprostasis and the very rare occurrence of diphtheric enteritis in its sequence does not support the view of constipation as an etiologic factor. In the chapter on Constipation I have already dwelt upon the fact that serious injury may be inflicted on the wall of the colon by fecal accumulation. Such injuries are, as a rule, caused by fecal tumors, and are usually limited to the descending colon or sigmoid. Such cases generally present the history of persistent chronic constipation. One often finds, in such cases, dilation of the colon and membranous colitis, these conditions being in my own experience more frequent consequences of obstipation than ulcers or colitis. Obstipation is so wide-spread a condition that it can not logically be advanced in explanation of endemic dysentery or of local epidemics in cities or special institutions. Constipation seems to be on the increase in our country, while dysentery and colitis are, according to my recent investigation of municipal statistics, on the decrease. Fecal accumulation, it is conceivable, may exert a traumatic influence upon the mucosa by compressing and bruising it, and thus lead to laceration and perhaps catarrhal ulcers. I do not wish to imply that these masses may not, in particular cases, induce even a colitis accompanied by a membranous exudation, but the local inflammation being once set up in this manner, the work of micro-organisms and the absorption of toxic substances are favored. The products of constipation may thus, in fact, give rise to a septic inoculation. From the facts stated, the conclusion is justifiable that constipation can not be a frequent cause of pseudomembranous enteritis.

Secondary Pseudomembranous Enteritis.

The diseases which may secondarily be followed by diphtheric enteritis are, in the first place, all those which can produce a severe cachexia: Chronic Bright's disease, heart affections, and the septic conditions, typhus and typhoid fever, cholera, variola, scarlatina, and, according to Bristow, it is frequently associated with pneumonia. Concerning the diphtheric colitis occurring in uremic conditions I refer to the chapter on Intestinal Ulcers of Uremic Origin.

The **pathology, diagnosis, symptomatology,** and **treatment** will be considered in the chapter on Dysentery.

5. MEMBRANOUS COLITIS OR ENTERITIS AND MUCOUS COLIC.

Definition.

A clinical combination of symptoms characterized by periodical colicky abdominal pains associated generally with abnormalities of the secretory and absorptive function, and with the discharge of peculiarly formed mucous masses, sometimes resembling exact casts of the intestine. It is a disease that has been very thoroughly investigated and described by American clinicians long before it met with the critical consideration of foreign physicians. According to

J. J. Woodward (*l. c.*, p. 363), it was first described in this country by Mason Good ("The Study of Medicine," cl. 1, ord. 1, species 7, vol. 1, Philadelphia, 1825, p. 162, "Diarrhœa Tubularis—Tubular Looseness"). It has also been described under the following synonyms: Infarctus (Kaempf); diarrhœa tubularis—tubular looseness (Good); follicular colonic dyspepsia; follicular duodenal dyspepsia (Todd); pellicular enteritis (Simpson); pseudomembranous enteritis (Cruveilhier); entérite pseudomembraneuse (Laboulbène); painful affection of the intestinal canal (Powell); mucous disease (Whitehead); hypochondriasis pituitosa (Fracassini); fibrinous diarrhea (Grantham); mucous disease of the colon (Clark); chronic, catarrhal, or mucous diarrhea; colique glaireuse, of the French; chronic exudative enteritis (Hutchinson); diarrhœa febrilis (Van Swieten); paraplexia rheumatica; chlorosis pituitosis; diarrhœa pituitosa (Sauvages); arthritis chlorotica (Musgrave); colica pituitosa (Sennertus); scelotyrbe pituitosa (Perywinger); mucositas intestinalis colloides; concretiones gelatiniformes intestinales (Laboulbène); tubular exudation casts of the intestines (Hutchinson); diarrhèe glutineuse; entérite glaireuse (Nonat); croup intestinal (Clamens); herpétide exfoliatrice (Gigot-Suard); colica mucosa (Nothnagel); côlite sèche (Potain); dysenterie du colon transverse (Lasègue); entérite mucino-membraneuse (G. Sée); chronic pseudomembranous gastro-enteritis; chronic pellicular inflammation of the intestinal mucous membrane; chronic croup of the intestines; mucous disease of the colon; chronic mucocolitis (Habershon).

For the first exact and classical description of the disease we are indebted to J. M. DaCosta ("Amer. Jour. of the Med. Sci.," October, 1871, p. 371). The most complete historical account of the literature relating to intestinal diseases associated with discharge of membranes or tubes composed entirely of mucus is found in the classical work of J. J. Woodward (*l. c.*, pp. 363–367). According to this account, the first one to describe this affection was Fernelius ("Patholog.," lib. VI, cap. 9, Paris, 1854, p. 181). This author described two cases, one of which proved fatal, and he recognized the true nature of the abnormal intestinal secretion, which he described as inspissated mucus. Morgagni (Epistle 31) has presented us with a masterly discussion of the various kinds of membranous substances which may be passed by the stool, and asserted that membranous substances may be passed either when there is dysentery or when there is not. He described three kinds of such membranes: (1) Gangrenous sloughs of the intestine; (2) false membranes; (3) sloughs with false membranes adhering to them.

The first modern publication of importance in Germany on this disease was given by E. von Leyden ("Deutsch. med. Wochenschr.," 1882, Bd. XVI u. XVII), which was followed by numerous very thorough reports on this subject, especially by Nothnagel ("Colica Mucosa," "Beiträge z. Physiol. u. Patholog. d. Darms," 16. Capitel, 1884), who emphasized that the nature of the disease was that of a mucous colic, and that an actual inflammatory enteritis need not exist. The first case of this kind which was examined at autopsy was reported by O. Rothmann (father) ("Deutsch. med. Wochenschr.," 1887, No. 27). It is not evident from the description of this author whether histological examinations were made. C. Ruge, who made the examination of the entire intestinal tract, states that nothing abnormal was found. M. Rothmann (son of the preceding) ("Zeitschr. f. klin. Med.," 1893, Bd. XXII) reported the autopsy of a case of membranous enteritis or colitis which was examined very carefully by histological methods, and gave every evidence of an inflammatory colitis. The assertion that Paulus Ægineta was familiar with membranous enteritis is doubtful—at least it could not be verified by J. J. Woodward (*l. c.*, p. 364, foot-note).

Nature and Concept.

There are three principal symptoms: (1) Painful intestinal contraction; (2) characteristic formation of mucous masses; (3) abnormalities of intestinal functions. The type of this disease which occurs most frequently and which is most characteristic is that in which an acute attack of abdominal pain is accompanied or followed by a profuse discharge of mucus in forms of membranes, tubes, or a homogeneous mass resembling liquid glass, glue, or frog spawn. These cases, as a rule, are not associated with any inflammatory process of the colon. I have personally examined eight such cases by H. A. Kelly's method of atmospheric distention of the rectum and sigmoid, and have not been able to ascertain that an inflammatory process was present in the parts capable of inspection. I have also attended two autopsies, one on a male and the other on a female subject, who had frequently been subject to attacks of this disease during life. Sections of the colon were examined microscopically from regions where a thick deposit of mucus had been superimposed upon the mucosa. I was not able to detect the histological evidences of inflammation in these sections, and for that reason incline to the view that the disease was unaccompanied by an actual colitis, thus confirming Nothnagel's view that mucous colic does exist in the absence of an inflammatory process in the colon and small intestine. His designation for this condition—" colica mucosa "—appears therefore a logical one. Osler (" Principles and Practice of Medicine," p. 396) has had opportunity of seeing these membranes *in situ*, closely adhering to the mucosa of the colon, but capable of separation without any lesion of the surface.

There is, however, a second type of this disease in which characteristic large masses of mucus are discharged, but the feature of colic and pain is not so pronounced; in fact, I have seen cases in which every stool was either followed or preceded by typical membranous discharges and the patient experienced no pain whatever. Krysinski (Inaugural Dissertation, Jena, 1884) describes six cases in which there was no pain, but simply a strong desire for stool or a necessity for evacuation. Evidently such cases can not be grouped under the heading of mucous colic, and indeed it has been found that they have arisen on the basis of an inflammatory enteritis. This is confirmed by the autopsy investigation of the younger Rothmann, and by two cases which I have examined personally at autopsies occurring in my service.

Sex.—The disease occurs most frequently in the female sex. According to Litten, 80 per cent. occur in women; according to Kitagawa, 90 per cent.; according to Einhorn, 90 per cent.; and according to my own cases—a total of 60, among which were 46 women and 14 men—76 per cent. occur among women. The prevalence of constipation, various types of enteroptosis, and uterine displacements have been cited in explanation of this predominance of the disease among the female sex.

Age.—The majority of cases occur between the ages of twenty and forty years. Longuet ("Réc. de mém. de méd. Milit.," 1878) and Ullmann (" Deutsch. med. Wochenschr.," Bd. XCIV, No. 2) have described cases in new-born children. Boas (*l. c.*, p. 243) has described a case in a very neurasthenic girl two years of age. J. Comby recognizes a distinct mucomembranous enteritis of young infants ("Traité de therapeutique," edited by Albert Robin, Fascicule XIII, p. 125). Löwenstein also described a number of cases occurring in early childhood ("Deutsch. med. Wochenschr.," Bd. LXXXIX, No. 2).

Etiology.

In the foregoing I have given in brief the histological reasons why I incline to the view of Nothnagel that mucous colic and membranous enteritis and colitis are two separate and distinct clinical entities. It is, therefore, reasonable to assume that the etiology must be different. However, the discussion among prominent specialists has not matured sufficiently to permit of the announcement of dogmatic statements concerning the causation of this disease. In the first classical description of membranous enteritis by our countryman, DaCosta (*l. c.*), he expressed the opinion that it was of neurotic origin; and later American clinicians—Max Einhorn (*l. c.*) and Walter Mendelson (*l. c.*)—still emphasize the neuropathic character of it. Mendelson asserts that if neurasthenic patients be closely questioned, very few will be found who have not had at one time repeated characteristic passages of stringy mucus associated with abdominal pains. Siredey was, I believe, the first who characterized membranous enteritis as a neurosis of secretion. The French writers, Albert Mathieu (*l. c.*), Potain (*l. c.*), and G. Sée (*l. c.*), assume the preexistence of a superficial inflammatory enteritis before a disease of the type of membranous enteritis can develop. Vanni, an Italian clinician (*l. c.*), is the only one who has more closely defined the process in question as a "myo-angioneurosis," with hypersecretion of mucus. In 1896 Dauber described a continuous secretion of mucus from the gastric mucosa for which he proposed the name of gastrosuccorrhœa mucosa, in analogy to the well-known gastrosuccorrhœa acida. This very rare condition, in which the secretion of mucus must be admitted as assuming the rôle of an independent pathological process, gives the clinical picture of a gastroblennorrhea. The evidences of pronounced gastritis were satisfactorily excluded. It is conceivable that there may be conditions of the mucosa of the colon in which it continuously forms mucus which condenses and solidifies on its surface, particularly when favored by a coexisting constipation (see Dauber, "Arch. f. Verdauungs-Krankheiten," Bd. II, S. 179).

Sven Åkerlund ("Arch. f. Verdauungs-Krankheiten," Bd. I, S. 421) regards this disease as a variety of enteritis or of chronic colitis, holding that the disease develops on the basis of an already existing or beginning catarrhal inflammation of the colon, under the exciting influence of one or more specific agencies, the nature of which he does not indicate. This view is essentially that of Krysinski (*l. c.*). Von Leyden conceives the process to be a croupous one, analogous to the croupous formations in certain types of bronchitis. The objection to this view is that the truly croupous or diphtheric colitis presents an entirely different clinical picture, such as I have described in the chapter devoted to that subject. Von Leube (*l. c.*) and Rosenheim (*l. c.*) regard this disease as a neurosis of secretion. Courtois-Suffit ("Traité de Médecine," "Publiée par Bouchard et Brissaud," tome IV, p. 423) favors the view of a superficial catarrhal lesion as a cause of the disease. A number of etiological factors have been conceded because of their constant, or at least frequent, coexistence with membranous colitis. Among these are principally habitual constipation and enteroptosis. Einhorn (*l. c.*) and Ewald (*l. c.*) have observed attacks of diarrhea in connection with enteritis. This must be a very exceptional occurrence, for in sixty clinical cases in which I have made a note of the state of the bowels I have observed only two in which diarrhea was marked. Glénard (*l. c.*), Ewald (*l. c.*), A. Mathieu (*l. c.*), Åkerlund (*l. c.*), Einhorn (*l. c.*), Boas (*l. c.*), and de Langenhagen (*l. c.*) emphasize a connection between displacements of the colon and membranous enteritis. When one reflects how frequently displacement of the colon may lead to constipation, the etiological connection seems well founded. Letcheff and Ozenne (*l. c.*) attribute some causative influence to disease and displacements of the uterus, which may also be explained by the tendency that displacements, adhesions, and disease of the uterus and ovaries have to causing constipation. Constipation, in my opinion, although a condition favoring the formation of mucous colic and enteritis, can not logically be considered a direct cause of it, because the latter diseases are rare in comparison to the great frequency of habitual constipation.

Chevalier (*l. c.*), A. Mathieu (*l. c.*), and de Langenhagen (*l. c.*) have called attention to an interesting connection between membranous enteritis and intestinal lithiasis. There is no doubt that excess of uric acid may, under certain conditions, find its way out through the intestinal mucous membrane. Sir Dyce Duckworth and Sir A. Garrod have described conditions of enteritis in gouty subjects which, in their opinion, were due to gout of the intestine. Hayem has recorded a case where enteritis occurred, the villi being strewn in small uratic incrustations, and in his "Treatise of Gout," page 89, Hayem asserts that severe colic, tympanitic distention, enteralgia, and diarrhea are sometimes distinctly referable to gouty disorder. Alexander Haig, of course, believes that a very large number of cases of intestinal indigestion leading to inflammation and putrefaction are really nothing more nor less than gout of the walls of the intestine ("Uric Acid in Causation of Disease, etc.," p. 330). Dieulafoy (*l. c.*), in speaking of intestinal lithiasis, has described definite periodical attacks of colic which are accompanied or followed by the formation and evacuation of considerable uric acid. He regards intestinal lithiasis as an expression of a gouty diathesis, and Mathieu holds that this condition occurs quite constantly in connection with membranous enteritis. From a large experience with intestinal troubles occurring in gouty patients, and after a critical consideration of the literature bearing on this subject, particularly the interesting forms of membranous enteritis which can be produced experimentally by certain inorganic and organic chemical bodies—for instance, nitrate of silver, copper, lead, and alum salts —which act in an analogous manner to uric acid, and from the great frequency of uric acid diathesis in just such neurotic, hysterical, and hypochondriacal subjects in which membranous enteritis has been most frequently observed, I can not free myself from the impression that, if not uric acid, perhaps other excretory products may have a causative influence in the production of this disease. I have frequently and consecutively examined the stools of two patients suffering from membranous colitis for uric acid during the attacks and in the intervals, and found quantities of uric acid which I failed to find in the evacuations of healthy individuals or of the same persons when they were free from these attacks. Even if we disregard the great importance which Sir Dyce Duckworth, Sir A. Garrod, and Alex. Haig attribute to intestinal uric acid infiltration and to the factor of uric acid in the intestinal lithiasis of Dieulafoy and Hayem, we must not overlook that while the uric acid itself may not be the causative factor, it can logically be regarded as the exponent of a large number of other toxins and injurious substances that seek their way out through the intestinal mucous membrane in disturbances of metabolism and renal insufficiency. The passage of such injurious substances through the walls of the intestines can readily be conceived to lead up to such a condition, producing hypersecretion of mucus with the accompanying characteristic symptoms of mucous colic and eventually even of enteritis membranacea. If chemical substances that are injected by irrigation—for instance, tannin, alum, and nitrate of silver—can produce membranous colitis, it is very probable that similar agents, when they are excreted through the intestinal mucosa, can produce an analogous condition. In the section on Diphtheric Dysentery I have cited the references giving the evidence that mercury, for instance, which has been used in the form of bichlorid of mercury for irrigating the uterus, had been the cause of a diphtheric dysentery. In this case and others the mercury had been absorbed from the uterine surface, and in other cases from the skin, and had been excreted through the intestinal wall. Even iron, given hypodermically, is secreted through the gastro-intestinal mucosa, and may cause digestive disturbances (L. Lewin, "Die Nebenwirkungen der Arzneimittel," 2. Aufl., Berlin, 1893). Haig (*l. c.*, p. 36) cites the evidences proving that zinc, copper, and lead are excreted through the intestinal tract and may give rise to a condition of colic with constipation or alternating constipation and diarrhea.

There is no doubt in my mind on the basis of quantitative analysis of stools previously mentioned that the waste-products of nitrogen metabolism in passing through the intestinal mucosa may exert a similar effect as these chemical agents and constitute a causative factor in the production of membranous colitis. In order to effect the exceptionally large mucous stools, two conditions are essential: (1) An excessive secretion of mucus from the intestinal epithelium. This may be due to a neurosis of secretion, as Leube, Vanni, and Siredey (*l. c.*) have pointed out, without the presence of any inflammatory changes in the mucosa; or it may be due to an actual enteritis or colitis. This mucus will not, however, be solidified and concentrated unless we have the second condition—viz. (2) stasis of the contents of the colon. Constipation, therefore, effects the condensation of the mucus on the surface of the intestine. There may, perhaps, be a third condition, and that is an altered chemical reaction of the contents of the colon. Mathias and Marquardsen ("Verhandlungen d. XVI. Congresses f. innere Medicin") have proved conclusively that the reaction of the intestinal contents in the ileum is alkaline, caused by the presence of carbonates and phosphates, but that the contents are saturated with carbonic acid. This therefore gives a simultaneous acid reaction to indicators sensitive to carbonic acid. This subject has been ventilated in the chapter on Examination of the Feces. Just here I wish to emphasize that these normal chemical reactions of the intestines favor the solubility of the mucus—that is, the small amount of it which is secreted normally. Should the chemical reaction change,—that is, should, for any reason, the alkalinity of the colon contents be reduced,—it would inevitably cause a deposition of mucus on the walls of the colon, and uric acid is a substance which in its passage through the intestinal wall would be capable of neutralizing the succus entericus and reducing the alkalinity of the intestinal mucosa and also of the contents.

The artificial production of membranous colitis by irrigations of the colon with nitrate of silver, tannin, alum, etc., should be a caution against the therapeutic use of these substances in this disease. I have repeatedly observed cases in which the discharge of tubular mucous casts of the intestine had been actually kept up by the means used to treat the disease.

Pathology.

The nature of the morbid process that constitutes membranous enteritis is capable of investigation from a number of standpoints. First, a study of the anatomical and histological evidences found at autopsies; second, experimental investigation on animals; third, analysis of the subjective and objective symptoms and signs during life; fourth, microscopic and chemical examination of the intestinal discharges ; fifth, comparison of the intestinal process with analogous occurrences in other mucous membranes.

1. I have studied histologically two cases of mucous colic in which I was unable to find the least microscopic evidence for the existence of an enteritis or colitis before death, and I have also had opportunity to study the small intestine and colon of two other cases of membranous enteritis which I had observed at intervals for six and ten years respectively. In these two cases there was the undoubted histological evidence of catarrhal inflammation of the ileum and colon in one case, and of the colon alone in another. I regret that a more extensive histological description of these two cases can not be given, because pathological investigations of the intestine of patients afflicted with this disease are very rare. If one disregards a report of autopsies in which no clinical observations were made during life,—and such cases are really worthless for our purpose,—we find in literature only two authentic cases : those of O. Rothmann (*l. c.*) and M. Rothmann (*l. c.*). The case of O. Rothmann had presented the typical clinical history of mucous colic. At autopsy a tear was discovered in the duodenum that had led to a fatal perforation peritonitis. The autopsy, which was made by C. Ruge, seems to have been a careful and thorough one, but it is stated that although the entire intestinal tract was thoroughly searched, nothing abnormal was found. The second anatomical report is by M. Rothmann, and was made on a female patient who died, in consequence of a carcinoma at the base of the skull, on November 25, 1892. Since June 14th of the same year she had suffered with obstinate constipation. On October 25th an irrigation of the colon was made and several meters of white, mucous, stringy masses were evacuated. The patient seemed to suffer no distress in consequence of this experience. The later history gives an account of repeated irrigation, with the same result—viz., the evacuation of similar masses without any fecal admixture and without pain. As the histological finding of this case corresponds very closely to one of my own observations, I feel justified in reporting it with greater detail. The descending colon, sigmoid flexure, and rectum contained quantities of membranous deposit which could be readily drawn from the congested mucosa without loss of substance. Wherever the colon was filled with these membranes it

appeared free from feces, but wherever there were no membranes the feces were present in abundance. This was particularly the case in the ascending colon. Throughout the entire colon the mucosa was reddened, injected, and hyperemic; especially so in the transverse colon, where it was also very strongly convoluted. The descending colon was also strongly contracted and the mucosa thrown into deep folds, between which whitish casts of the free spaces still maintained within the colon, in spite of the contraction found. The deposits were partly broad membranes, or in form of string-like casts of the free spaces. In the small intestine—the ileum—there was considerable fecal matter, and the mucosa was slightly reddened. Histological examination by various staining methods gave the evidence that the membranes, etc., were composed of mucin and did not contain fibrin. The masses of mucus had completely replaced the epithelial covering of the mucosa, and had forced themselves into the lumen of the glands, extending to the fundus. Lateral extensions of the masses could be traced into the beaker cells of the glands. The interglandular spaces were distended by decided small-cell infiltration and immigration of other cellular elements, and there was a distinct broadening of the entire mucosa. In this case there are, therefore, the unmistakable evidences of a catarrh of the colon and also of the ileum. There are, therefore, certainly two types of this disease, as demonstrated by the pathological evidences so far obtainable: The first type presents the clinical picture of mucous colic (colica mucosa) with a perfectly intact intestinal mucosa. The second type presents an augmented evacuation of membranous masses as a result of a coexisting enteritis or colitis. These two conditions have the enormous evacuation of mucus in common.

2. The experimental investigations that have so far been made with a view to clearing up the pathology and etiology of the disease (Vanni, *l. c.*, and Sven Åkerlund, *l. c.*) have led to no results.

3. The analysis of the subjective and objective physical signs and symptoms—that is, the clinical picture—is, after all, the most important feature in the study of this disease, and without this feature pathological reports have no value whatever. In a critical analysis of the symptomatology one can not fail to be struck with the marked divergence that it presents from actual enteritis or coli-

tis. The statistics that I have given demonstrate that the disease, or at least one type of it, attacks principally neurotic women, whereby it decidedly differs from enteritis. In the great majority of cases we are confronted with a paroxysmal attack, characterized by a colicky pain located in the transverse and the descending colon, and by the discharge of masses of mucus. The pain is, in my opinion, a secondary effect, provoked by the excessive accumulation of mucus in a reflex manner, just as gaseous distention may cause tetanic contraction of the colon in flatulent colic. In the intervals between the attacks the patient does not suffer, and, though constipated, has evacuations of normal composition or consistency, at least so far as mucus is concerned. Right here I wish to emphasize that it is not demonstrated that the stools of such patients are entirely normal in the intervals. It will require long and careful investigation by chemical analysis of the stools before this can be proved. In eleven cases of membranous colitis examined by myself the amount of the total ethereal and preformed sulphates was very high, and there was an excess of indican in the urine, suggesting intestinal autointoxication. As the normal stools contain no appreciable amount of mucus, it has been urged that the presence of an excessive amount of mucus, as has been emphasized in the section on Enteritis, is an evidence of the presence of intestinal catarrh. This deduction is correct in the main, but Nothnagel, in formulating and maintaining it, admits also the possibility of the production of enormous masses of mucus even without catarrh; and mucous colic and dystrypsia are the conditions in which this is possible (Nothnagel, *l. c.*, p. 17).

A consideration of the facts thus far stated can not fail to bring the conviction that mucous colic and colitis membranacea constitute a disease in themselves. Both these conditions may occur without any evidences of inflammation within the intestinal mucosa; on the other hand, there can be no doubt that copious discharges of membranous, tubular, or unformed mucus may occur in the course of actual intestinal or colon catarrh. But these cases run their course without the characteristic sign of paroxysmal painful colic. Such cases have been described by Krysinski (*l. c.*) and M. Rothmann (*l. c.*). A similar case, in which undoubted enterocolitis had existed for ten years, has occurred in my private practice. The patient, a female thirty-six years old, had been constipated since her eighteenth year, but for the last ten years constipation had alternated with profuse diarrhea, both kinds of passages showing large quantities of mucus, which was, as a rule, formless, like frog spawn, or in long strings. Present history: Once or twice every year she has periods in which her bowels act apparently normal as regards consistency and number of movements—that is, there is one formed passage in twenty-four hours. During this period of apparently normal intestinal action she will generally have evacuations which consist entirely of mucus, presenting the form of tubes, varying from ten centimeters to half a meter in length. Much of the mucus appears at first as broad bands, membranes, or ribbons, but when they are floated out in formalin water, it is generally discovered that they are tubes. Direct inspection of the rectum and sigmoid by atmospheric distention and the long sigmoidoscope gives evidence of a chronic sigmoiditis, but in the membranes the histological evidences of chronic catarrhal inflammation are also found. Such cases of inflammatory catarrh of the intestine are really deserving of the name of membranous enteritis. It must be borne in mind, however, that they are extremely rare, and that the majority of cases of this disease do not present the histological evidences of an inflammatory process, and the designation of Leube, "secretory neurosis," can not be objected to on valid grounds as a term applicable to the majority of cases of this disease.

4. Microscopic and Chemical Examination of the Intestinal Discharges.—The best preservative for keeping the membranous tubular or homogeneous mucous masses is a 10 per cent. solution of carbolic acid in water or a 5 per cent. solution of formalin. The latter I have found objectionable because it produces irritation of the eyes. Alcohol destroys certain typical microscopic peculiarities of the mucus, and preparations placed in solutions of corrosive sublimate are useless. There are three forms in which the mucus is generally found in the evacuations: (*a*) The formless, homogeneous masses of mucus resembling frog spawn, egg-white, or liquid glue. They may be colorless or exhibit a brownish color due to admixture of fecal constituents. Mucus of this character is usually mixed with feces, the feces coming out but slightly mixed with the mucus and then followed by a huge mass of this frog-spawn-like material. (*b*) Here the mucus presents a cylindrical or tubular form, which may show considerable toughness and consistence. The tubes are opaque, grayish white, and at times mixed with the feces. Their length varies from ten to fifty centimeters. Occasionally they make up the entire bulk of the evacuation. (*c*) This form includes all the configurations in which the evacuated mucus assumes the shape of membranes, ribbons, or strings. The membranes generally occur in small fragments, and they are best recognized when floated out or suspended in physiological salt solution. This classification is, however, entirely schematic, and does not mean to exclude the possibility that formations of all three categories can occur simultaneously. Thus, membranous or tubular formations are frequently accompanied by larger or smaller quantities of formless mucus; occasionally the membranous and tubular types present a variety of transitional forms in the same evacuation. Blood is a rare admixture, but when it does occur on the surface of membranes or tubes, it is significant of an intimate adhesion of the mucous substance to the tissue of the portion of the colon from which it has been detached. The presence of blood and cylindrical epithelial cells does not necessarily indicate inflammatory catarrh. Such epithelium may be present in the membranous fragments in enormous numbers. The cylindrical epithelium can, even under normal conditions, be very readily detached from the mucosa of the intestine. The mucous deposit is frequently strongly adherent to the surface of the mucosa, and the violent intestinal contractions which occur during the paroxysms of colic are sufficient to detach this epithelium mechanically.

The substance, which is designated generally as mucin, is, according to our present conception, not a uniform body from a chemical standpoint. Generally only such substances are included under that term as present the physical properties of ordinary mucus. The mucin reactions of Hammarsten frequently result in extremely varying products. In order to stain mucus for the detection of the histological elements contained in it, as well as of the ground substance itself, thionin as first used by Hoyer (*l. c.*) is the most serviceable substance. When the membranes from this type of colitis are given varying chemical reaction by the addition of decinormal solutions of KOH or HCl, thionin acts in varying ways. If the mucin is neutral, thionin will stain it red-violet, but any tissue or cellular elements, as well as other secretions, will be stained blue. If the solution of mucin is made alkaline, the mucin, as well as all other substances, assume a pink color. If the solution is made to react distinctly acid, or if it is already so,—this may be caused, for instance, by the gastric secretion,—then all the cellular elements as well as the mucus will stain blue. If transverse sections of the intestine are made and it is desired to stain them, together with the adherent deposit of condensed mucus, very good preparations can be obtained by Weigert's fibrin stain. Of other stains, methyl-blue, safranin, and eosin can be used with success. Safranin stains the formed constituents of the membranes, but hardly tinges the ground substance at all. The more the ground substance can be freed from fecal admixture, the more successful will the preparation be. In well-stained specimens, under low magnifying power, we then observe a hyaline homogeneous ground substance in which numerous smaller and larger stained points are observable. Under higher powers a striation is observable in the ground substance, and the points before observed can be made out as stained cells. The formed elements are composed: (1) Of epithelial cells; (2) of red blood-corpuscles; (3) of leukocytes; (4) of bacteria; (5) of lower animal organisms, which are very rare; (6) among the rare findings in these membranes are crystals of cholesterin, Charcot-Leyden crystals, crystals of inorganic salts, fat, and the ordinary admixtures which occur in fecal matter. Fresh and unchanged cells are very rarely met with; the majority of them are in a state of necrobiosis. Kitagawa (*l. c.*) considers this degenerative process in the cells a coagulation necrosis. Nothnagel (*l. c.*) holds that they are the consequence of desiccation, while Adolph Schmidt (*l. c.*) finds the cause of this degeneration in an inbibition of the cells with fatty soap. After the removal of this inbibition the cells resume the shining, transparent appearance of normal fresh cells.

From the microscopic appearance of the dejecta, therefore, we can elicit no evidences upon which the conception of an inflammatory or noninflammatory origin of the mucus could be based. It was thought that the absence of round cells and pus-cells, which has been confirmed by a number of reliable observers, argued against an inflammatory process. However, these cells are generally absent also in the mucous discharges of catarrhal enteritis. If round cells would be found in large numbers in any case, it would, according to Nothnagel, speak for the presence of an inflammatory catarrh. Cup or beaker cells have rarely been observed, and the origin of the other degenerative forms of which we have spoken has not been sufficiently cleared up to permit of a decision on the question of the inflammatory or noninflammatory origin of the mucus in any particular case.

5. Comparison and Analogy of the Intestinal Process with Similar Occurrences in Other Mucous Membranes.—Membranous colitis has been compared to chronic croupous bronchitis, and, indeed, there seems to be a certain parallelism between the two, for it is generally believed at present that the bronchial casts in this disease are not composed of fibrin, but, according to Neelsen and Beschorner, they are composed entirely of condensed mucus. Klein holds a similar view. The mucous membrane of the bronchial tubes in this disease shows no anatomical changes, according to Litten. The disease has also been compared to membranous dysmenorrhea by Wernich. The result of these analogous comparisons speaks against the inflammatory character of membranous enteritis and colitis, as well as against the fibrinous nature of the discharges, and argues in favor of the view which interprets the disease as a secretory neurosis. It is conceivable that such a secretory neurosis may be superadded to an already existing inflammatory catarrh, but it would be difficult to decide in such cases whether we are dealing with a simple enteritis or colitis with excessive production of mucus, or the same anatomical conditions to which a secretory neurosis might have been superadded.

Complications.

The most frequent complications are those which have been mentioned under Etiology. These are, to recapitulate, various forms of enteroptosis, nephroptosis, gastroptosis, and coloptosis, intestinal lithiasis, diseases of the uterus and adnexa, and insomnia. Courtois-Suffit has observed elevation of temperature; Richardière, albuminuria; Cantru, epileptic attacks. Einhorn has observed achylia gastrica in five out of twelve of his cases. Other rarer complications are intermittent heart's action, tachycardia, dyspnea, somnolence, amblyopia, convulsions in children, neuralgia, melancholia, and general nervous tremor.

Symptomatology.

Patients of this type have been suffering with constipation for a long time before the typical attack of enteritis develops. This is preceded by frequent pains, as a rule, localized in the transverse or the descending colon. The pains gradually become aggravated, and sometimes become so intense that the patient has a sensation of impending dissolution. When a patient has never had these attacks before, the symptom of pain may give the impression of a patient suffering from the passage of gall-stones or renal calculi. I remember particularly one case, in the wife of a brewer, where the pains, of a very intense character, existed for four days, and the passages produced by calomel and castor oil did not contain any mucus. On the fifth day the trained nurse exhibited to me an almost perfect cast of the colon, twenty-two inches long, which she had suspended in a large wide glass jar full of water. The diagnosis can not be made until the evidence of membranous formation is presented. Membranous enteritis might exist without pain, but it can not be said to exist without the characteristic mucous discharges just described. The discharge of the membranes may, at times, be painless. In other cases very painful attacks may vary with painless ones. The other symptoms are referable to the nervous condition of the patient, and constitute only partial or accompanying phenomena or complications. There are few objective symptoms of importance. Perhaps the most constant is the great sensitiveness to pressure localized in the colon, particularly the descending colon and sigmoid flexure. This sensitiveness may disappear entirely as the case improves. The most important objective sign is the discharge of the characteristic membranous, tubular, or unformed mucous masses, consisting almost exclusively of mucin and a proteid body. The amount of the latter varies according to the number of cellular elements contained in the mucus. The discharges contain no fibrin. The majority of observers—Åkerlund, Nothnagel, Kitagawa, Rothmann, Ad. Schmidt, Boas, and others—agree on this point. A few observers have found fibrin present in traces, and Germain Sée holds that this is a particularly important sign, since he bases upon it the conclusion that the curious discharges could not be of inflammatory origin.

Diagnosis.

After the detailed account which I have given, it will hardly be necessary to dwell extensively on the diagnosis. In all cases of doubtful pain in the abdomen in neurasthenic females exploratory lavage of the colon should be undertaken, when the characteristic membranous mass will be detected. Nothnagel suggests that the membranes might be mistaken for fasciæ and tendons, as well as arterial and venous tubules taken in the food. This I consider extremely doubtful. In the same way the supporting tubular fibers of the orange could hardly be confounded for the structures of membranous enteritis, particularly if they are examined with the microscope. Nothnagel also mentions a confusion that occurred the first time he observed a case suspected as membranous enteritis, when he obtained a substance thought to be a characteristic discharge, but microscopic and chemical investigation showed it to be a milk coagulum. Membranous enteritis, as I have pointed out, may be produced artificially, and I have been called into consultation in two cases where there was no doubt that the physician himself had caused this disease, or at least brought on the characteristic tubular mucous casts by irrigations with nitrate of silver. The disease, which had continued uninterruptedly for six weeks in one case, ceased entirely when the irrigations were discontinued. There are cases of actual inflammatory colitis or enteritis associated with the evacuation of excessive masses of mucus. If these masses assume the shape of membranes or tubes, a diagnostic differentiation will be difficult, but of little therapeutic consequence, as colitis presenting the foregoing symptom will have to be treated like colitis membranacea.

Prognosis.

In some cases the disease disappears entirely after a few typical attacks, never to recur again, but this is a rare experience. In the majority of cases the disease runs a chronic course, with remissions and intermissions. The prognosis is good so far as life, but is doubtful so far as restitution to integrity, is concerned.

Treatment.

An interesting discussion has developed in the literature of this disease, as to whether it should be treated along the lines of an enteritis or as an intestinal secretory neurosis. Nothnagel (*l. c.*, p. 147) favors a treatment directed toward the neurotic condition, and asserts that all therapeutic methods that have been directed against the supposed catarrh have shown rather poor results. According to Boas, however (*l. c.*, p. 247), great emphasis is laid upon a treatment of the enteritis. Einhorn (" Diseases of the Intestines," New York, 1900) classes the membranous enteritis among the secretory neuroses (p. 335), and does not consider it in the same chapter with enteritis and colitis. Disregarding this discussion for the present, membranous enteritis comes before the practitioner in two forms: (1) In the form of the acute attack, in which the most urgent indications are the relief of pain and prompt evacuation of the bowels; (2) in the form of the chronic stage, generally in the intervals between the painful attacks. The most important indications for treatment are the constipation, the enteritis or neurosis as the case may be, sometimes both, and the prevention of intestinal autointoxication. In the acute attack the pain is best combated by suppositories containing codein, belladonna, or morphin. Opium must be avoided because it increases the constipation. When the pain is so severe that there is no time to wait for the action of a suppository, one should not hesitate to give a hypodermic injection of ¼ of a grain of morphin at once, and place hot cataplasms upon the entire abdomen. Towels wrung out of very hot water and sprinkled with turpentine are quite serviceable. Heated thin bags filled with salt or hot-water bottles will answer admirably. In my experience infusion of camomile flowers and marshmallow-root (*Radix althæa*), which are recommended by Sven Åkerlund (*l. c.*, p. 423), are not capable of allaying the pain of the acute attack, but they may be of use in the form of large colon irrigations in the intervals between the attacks, when their emollient and demulcent effect is sometimes sufficient to allay the slight irritation and soreness that remains occasionally as quite an annoying feature for weeks. The best results are obtained by dietetic management and local treatment with irrigations of the colon. The treatment of the enteritis or the neurasthenia, whichever is most pronounced, can properly be undertaken either in conjunction with or after these most prominent indications have been fulfilled.

The Diet.—Von Noorden (*l. c.*), in 1898, ordered a diet rich in dross, shells, and fine seed, such as those contained in the blackberry, strawberry, and figs. In addition to this an abundance of fats, butter, and bacon. He recommends half a pound of coarse bread, rich in the shells of the cereal grain, like Graham bread, a day, and such garden and leguminous vegetables as are rich in cellulose; berries, such as currants and gooseberries, and grapes—in short, a diet rich in indigestible material, in order that it may form a considerable amount of chaff and dross for the intestines. He reported seven permanent cures and seven improvements in fifteen patients, while one case was unchanged. While this diet is theoretically correct, I have never found it practical to enforce it successfully. None but the most determined and enthusiastic patient is willing to submit to this coarse food, as some of them have called it. In addition to that, the diet seems to me actually contraindicated if there should be an enteritis or irritative condition in the intestine. Most authors of repute—Nothnagel, A. Mathieu, A. Schmidt, Ewald, and Germain Sée—distinguish carefully between an actual catarrh and the mucous colic proper that occurs with an intact intestinal mucosa in their considerations of enteritis membranacea. Von Noorden's diet undoubtedly is useful in simple atonic constipation. For the treatment of the constipation accompanying membranous enteritis, however, I prefer a diet which, while it adheres to the principle of von Noorden, does not include so much of shells and seeds to make it irritating. The diet-list and general food directions given in the chapter on Constipation will suffice for guiding lines for this purpose. The menu should be ample, containing a variety of meats, carbohydrates, and plenty of vegetables, as well as of butter and rich gravies. The object we set before us must be not only to cure the constipation, but to improve the general nutrition of the patient. The diet will, however, as a rule, not be sufficient to cure the constipation in these cases. The other physical methods mentioned in the chapter on Constipation may become necessary—for instance, electricity, massage of the colon by the cannon-ball method, and enteroclysis. It is important to avoid the use of purgative drugs, but if absolutely needed, the least harmful are aloes, tamarinds, castor oil, licorice powder, cascara sagrada, and rhubarb.

Treatment of the Constipation and Irrigation of the Colon.—While colon irrigations have for their object the simple removal of the accumulations of mucus, it is evident that if any inflammatory condition should exist in the colon, the method is an admirable one for local treatment. There are no specifics for the cure of this disease. If any method has yielded especially good results in my hands, it is the application of large oil enemata, according to the suggestion of Kussmaul and Fleiner—about 250 to 400 c.c. of pure olive oil, which should be washed by shaking it with water beforehand, in order to remove all possible traces of fatty acids, which are soluble in water. The oil should be warmed to body-temperature, and allowed to run slowly into the colon. The patient must be instructed to retain the oil in the bowel during the entire night, these oil enemata being usually given about 9 P. M. The patients, as a rule, succeed in retaining this amount of oil without being compelled to evacuate it during the night. If this quantity of oil is repeatedly ejected, a smaller quantity of oil should be used next time—perhaps not more than 150 c.c. These Fleiner enemata should be given daily for three weeks; thereafter every alternate day for three more weeks; and, finally, twice a week for one month. I generally advise, even after this period, one oil enema a week for six months. I have treated ten cases of membranous enteritis in this way from 1891 to 1896, and consider the treatment efficacious, because of this number eight have never had a return of their former disease, nor even of the constipation. The treatment was, of course, combined with diet and abdominal massage, which may have contributed largely to the results. Einhorn explains the action of the oil in part by assuming that the presence of the oil in the intestine prevents it from being left in an empty condition during the night, and thereby a spasmodic contraction is avoided. In addition to the oil enemata, the best cleansing enemata for the removal of the accumulated mucous masses is normal salt solution. I usually add to this three grams of bicarbonate of soda to the liter of normal salt solution. It may not effect a solution of the enormous masses of mucus, but it certainly facilitates their detachment from the location in which they are fixed, and also reduces the intestinal autointoxication. Many of the neurasthenic symptoms observed in this disease have recently been attributed to the absorption of toxic substances from the intestine, and the internal administration of bismuth salicylate and beta-naphthol has been recommended by some authors to counteract this effect. I have tried this and other so-called intestinal disinfectants in this disease, but have discarded them because I became convinced that they were no more effective than the irrigations with normal salt solutions alone. The addition of any other chemical substances to the irrigating fluid with the view of exerting an astringent or antiseptic effect—nitrate of silver, alum, tannin, salicylic acid, etc.—is dangerous. I have already narrated my experiences with reference to the effect of these substances in this condition.

The treatment of the neuroses may require hydrotherapeutic, electrical, and climatic measures, and in rare cases sanatorium treatment. The use of mineral waters has been of no practical utility in my experience.

The Management of the Complications.—Enteroptosis, gastroptosis, nephroptosis, uric acid diathesis, and affections of the uterus and adnexa may require special efforts directed to their removal or alleviation. F. Francke (*l. c.*), also Hale White and Golding Bird (*l. c.*), claim to have cured membranous colitis by producing an artificial anus, which served as the ending of the ileum, and kept the colon entirely free from food. When pronounced atony of the colon is unmistakably present, a prolonged course of treatment with strychnin sulphate, $\frac{1}{30}$ of a grain three times daily, has proved a valuable adjunct to the treatment. The same can be said of the use of dilute HCl in those cases in which the analysis of test-meals has demonstrated the coexistence of achylia gastrica.

SIGMOIDITIS AND PERICOLITIS.

Definition.—Inflammations in the left iliac region.

The sigmoid flexure, because of its characteristic anatomical construction, its location, and its function, is more frequently subject to catarrhal inflammation than any other part of the colon. The fecal mass as it enters the large intestine travels at a fairly uniform rate through the ascending and transverse colon. When it reaches the descending colon, the rate of propagation of the colon contents is slower than in the transverse or ascending colon. This is due to two factors. One would suppose that the rate of progress of the fecal matter in the colon would be faster in the descending colon than in the ascending and transverse colon, because it has gravity in its favor after it has passed the splenic flexure; but the rate is impeded by the peculiar corkscrew and twisted shape of the sigmoid flexure, and by the fact that the watery constituents of the fecal mass becoming absorbed in the ascending and transverse colon renders the contents drier and more difficult of onward propulsion. If the fecal mass is not at once evacuated after it has passed through the sigmoid, it is lifted back into the flexure by a retroperistaltic or antiperistaltic movement, and here it remains, becoming drier and drier, and, by its mere presence, acting as an irritant.

The name sigmoiditis has been applied to an inflammation of the mucosa of the sigmoid flexure which occasionally may extend to the peritoneum and the adjacent pericolic cellular tissue. This condition has been designated as "ileac phlegmon." The disease was first described by A. Mayor ("Revue Médec. de la Suisse Romande," 1893, No. 7, p. 421).

Etiology.

The disease was attributed by A. Mayor to constipation, retention of stool in the sigmoid flexure, and other irregularities of defecation. In one of the cases observed by me there was an obstruction in the rectum caused by a rectal valve of unusual dimensions, after the removal of which the disease disappeared spontaneously. It is to Dr. Thomas C. Martin, of Cleveland, O., that we are indebted for new and important knowledge concerning the normal and pathological function of these rectal valves ("New Evidence that the Rectal Valve is an Anatomical Fact," Louisville, John P. Morton & Co., 1896). In another case observed by me the stasis of the contents of the sigmoid was caused by a large uterine myoma, which, together with the uterus, had become retroverted upon the rectum, and firmly adherent. This case also was cured of its sigmoiditis after removal of the myoma. A third case was due to the frequent occurrence of a distended bladder, caused in turn by an enormously hypertrophied prostate gland.

This case was still under observation at the time of this publication, and was improving mainly because the bladder is kept empty by catheterization. In my opinion simple obstruction is not a sufficient etiological explanation. In one case which I could inspect with the sigmoidoscope numerous small erosions were visible to the naked eye, and it is probable that in other cases old or recent infections play an important etiological rôle. One of the cases described by Mayor had had dysentery for two years. It is much to the credit of this clinician, however, to have called attention to sharply localized and circumscribed inflammation in the left iliac fossa. The attention of medical men has been restricted to the right iliac fossa in the majority of patients that complain of pain in the lower abdomen, so that careful examination of the left iliac fossa is frequently omitted.

Sigmoiditis may be acute or chronic. The following will suffice for a synopsis of the four cases described by Mayor:

First Case.—A female, thirty-two years of age. Prolonged constipation; otherwise healthy. On examination the sigmoid flexure presents a cylindrical thickening, which can be made out as continuous with the descending colon above and extending into the pelvis below. The patient at no time had fever. Examination of the rectum, negative. The patient recovered, the main treatment consisting of rest and hot cataplasms.

Second Case.—A boy, fourteen years old, was attacked with severe pains in the left side; the sigmoid flexure was thickened and palpable as a swollen tube. After several days of fever the case recovered spontaneously.

Third Case.—A boy, eleven years of age, was attacked with pain in the left side, high fever, formation of abscess, and perforation of the abscess into the intestine.

Fourth Case.—A physician, forty-two years of age, was attacked with violent colicky pains in the region of the sigmoid flexure, accompanied by nausea, but without a previous history of constipation. No fever. The sigmoid was hard, swollen, and sensitive to pressure.

Galliard ("Traité de Médecine," Brouardel-Gilbert, tome IV, p. 603, 1897; "Gaz. des Hôpitaux," 1897, No. 7) reported a similar case which presented violent pains in the left iliac fossa, and severe general symptoms, together with fever. A tumor as large as an orange formed, and did not disappear until after three weeks. In the chapter on Appendicitis I will describe the abnormality where the cecum and the appendix may be displaced or distorted toward the left iliac fossa; naturally, under such a condition a diagnosis would be very difficult.

Diagnosis.

The disease has not been described with sufficient frequency or accuracy to permit of the formulation of diagnostic factors. It may be confounded with inflammation of the tube and ovary on the left side, with acute exacerbations occurring in the course of chronic enteritis, and with displacements of the cecum and appendix to the left side, as described in the preceding. All these mistakes may be avoided by careful inspection of the rectum and sigmoid, executed by the method of Thomas C. Martin, as described in the section on Rectal Diseases. Digital examination of the rectum or inspection through the proctoscope reveals very little of this disease. But the long sigmoidoscope, if used under anesthesia, has, in my experience, always to a degree disclosed the condition of the sigmoid flexure.

Pathology.

Nothing reliable is known concerning the pathology and pathogenesis of this disease, because results of autopsies concerning it have so far not been published in the literature of the subject.

Treatment.

The treatment is local and general. The constipation must be remedied at once, and the sigmoid flexure cleaned out. Of internal remedies, I favor castor oil in tablespoonful doses, until the bowel is thoroughly evacuated. The diet should consist of finely scraped beef, soft-boiled eggs, or simply egg-albumen, gelatin, and very thoroughly boiled rice or farina. Milk, which is an excellent article of diet in some intestinal diseases, has in two of my cases given rise to such firm and solid stools as to exert great pressure upon the inflamed portion of the sigmoid in their passage.

Externally, ice-bags or cold applications are useful at the very onset of the disease, especially if there is high fever, but after four days, if the induration persists or grows larger, hot cataplasms are in order. If the patient can stand it and it does not cause too much pain, local lavage of the sigmoid with saturated solutions of thymol have, in my experience, seemed to cut the disease short. After lavage in this manner I prefer to inject, under gentle pressure, about 200 c.c. of olive oil, to which about a teaspoonful of ichthyol has been added. In all cases in which there is severe pain morphin subcutaneously or in suppositories should be given.

Chronic Sigmoiditis.

There is a chronic localized inflammation of the sigmoid flexure in which the pain is permanently confined to the region of this part of the colon. There may be diarrhea or constipation. If the stools are liquid or semisolid, there is a strong admixture of mucus. If there is diarrhea, the evacuations are enveloped in mucus. The following is a clinical report of one of the cases that occurred in my practice.

Mrs. G. G., aged forty, has had six labors, all more or less difficult; the first three required the use of the forceps. In the last the perineum was torn through to the rectum. Eight years ago perineorrhaphy was successfully performed. Two years after the following symptoms gradually developed: Great distention and soreness in the left lower abdomen; for the first year to year and a half, constipation, but in the last year diarrhea, the discharge containing much mucus. She also complained of tenesmus. In the last year the diarrhea occasionally alternated with normal stools, but during the last three months constipation, diarrhea, and normal stools alternate in an irregular manner. Physical examination: Heart and lungs normal; right kidney slightly movable. Abdomen relaxed and pendulous. On distending the stomach with air the greater curvature is seen to be at the level of the umbilicus. Urine shows an excess of indican, but otherwise is normal. Palpation of the region of the sigmoid flexure elicits extreme sensitiveness. Examination of the rectum discloses no special pathological condition and no hemorrhoids, but about 3 1/3 inches from the anus a large spherical mass compresses the rectum, and, on palpation, gives rise to pain. By bimanual palpation, one hand in the vagina, this mass is made out as a uterine tumor of considerable size. In the knee-chest position the rectum and sigmoid are distended very satisfactorily by atmospheric pressure (see illustration in article by Thomas C. Martin). The mucosa of the upper rectum and sigmoid is congested, thickened, and thrown into numerous folds, which apparently can not be effaced by the distention nor by wiping across them with a ball of absorbent cotton held in a long pair of forceps. Here and there the mucous membrane is covered by adherent whitish or yellowish mucus. The end of the tumefied condition of the sigmoid toward the splenic flexure can not be made out.

Examination of the stool showed the absence of pus, amebæ or other parasites, and blood; but whether constipated or diarrheic, they always showed an excess of mucus. The patient has been wearing a large horseshoe-shaped pessary, which, when pressed down by the myoma, could be felt as a firm mass by the finger palpating in the rectum. The gastric peristalsis and secretion were normal.

Treatment.—Local irrigation with warm water; internally, castor oil. Diet as described previously. Rest in bed. Hot fomentations to the left iliac fossa. Recovery after one month of treatment, at which time the sigmoid was not sensitive to pressure and the stools appeared normal.

In connection with this disease I wish to emphasize that diarrheic stools containing much mucus may immediately precede a perfectly normal stool. This indicates that the diarrheic mucous stool is formed in the lower bowel, which, when being cleared out, gives way to a normal passage which has formed above the sigmoid, suggesting that the bowel above the sigmoid flexure is in a normal condition.

The Anus as a Portal of Entry for Infection.

There is no doubt in my mind that there are cases in which the infection seems to enter first at the anus. Dr. Stewart Eldridge, Assistant Surgeon in the United States Marine Hospital Service, has emphasized such an origin of Japanese dysentery in the Public Health Reports published by the United States Marine

Hospital Service, volume xv, No. 1, page 5. An epidemic and highly infectious gangrenous disease of the rectum, which may or may not be closely allied to dysentery, has been described by Sigaud ("Du Climat et des Malad. du Brezil," Paris, 1844) and Brett ("Indian Tribes of Guiana," London, 1868) as occurring in South America; and by Corney ("Trans. Epidemiological Soc.," 1887–88) as a serious cause of death in Fiji and other South Sea islands (see Davidson, "Hygiene and Diseases of Warm Climates," Edinburgh, 1893). This malady, as a rule, begins at the anus itself, which immediately becomes dilated and progresses rapidly upward until death occurs. I have met with other and more recent allusions to this interesting form of rectal disease, but am, at present, unable to refer to them. It is of interest in connection with this form of dysentery and the presumed method of infection which I have mentioned above (Eldridge, *l. c.*).

In the privies of large institutions, particularly in military institutions, in which the opening in the seats is much larger than can be covered by the buttocks, there is often a strong upward draft of air during the occupation of the closets. This would favor the impact of any bacteria in the air against the anus, particularly if paper were used which had remained for a long time in the privy.

Diagnosis.—The same factors that have been considered in the diagnosis of the acute form pertain also to the chronic type. In establishing the existence of the disease it is necessary to seek for the evidences of a colitis and marked sensitiveness localized to the sigmoid flexure.

The treatment is the same as that of chronic colitis given elsewhere.

LITERATURE.

1. Aegineta, Paulus, cit. by Da Costa, "Amer. Jour. of Med. Sci.," 1871, p. 321.
2. Åkerlund, "Arch. f. Verdauungs-Krankheiten," Bd. 1, S. 396, 1895.
3. Ashton, Lawrence, "Gastro-intestinal Catarrh," "Texas Clinic," Dallas, Texas, June, 1900.
4. Bellfrage und Hedenius, cit. from Virchow-Hirsch's "Jahresbericht f. 1876."
5. Bettmann, H. W., "Some Unusual Types of Gastro-intestinal Disorders," "Cincinnati Lancet-Clinic," June 23, 1900.
6. Boas, "Deutsche med. Wochenschr.," 1893, No. 41.
7. Boas, "Diagnostik und Therapie der Darmkrankheiten."

8. Boas, J., "Krankheiten des Darms," Bd. II, S. 513.

9. Bouchard, "Leçons sur les Autointoxications," Paris, 1887.

10. Bret, de, "Contribution à l'étude des concrétions muqueuses membraniformes de l'intestin," "Annal. de dermat.," 1869, No. 3.

11. Brunner, Wladislas, "Revue de médecine," 1892, p. 733.

12. Cautru, "Complications nerveuses de l'entérite mucomembraneuse—épilepsie, chorée," "Méd. moderne," Janv., 1895.

13. Chevalier, "Contribution à l'étude de la Lithiase intestinale," Paris, 1898.

14. Cornil, cit. from Siredey.

15. Courtois-Suffit, "Traité de médecine," by Bouchard et Brissaud, tome IV.

16. Courval, "Note pour servir à l'étude des concrètions muqueuses membraniformes de l'intestin," "Union méd.," 1869, No. 37.

17. Cruveilhier, "Anat. Path. gén.," tome II.

18. DaCosta, "Membranous Enteritis," "Amer. Jour. of the Med Sci.," 1871, p. 321.

19. Delafield, F., "Medical Record," May 11, 1895.

20. Dieulafoy, "Presse méd.," 1895, 10 Mars, "Acad. médic.," 1897, 23 Mars.

21. Dulin, C. W., "Gastro-intestinal Disease," "Kansas City Medical Index-Lancet," June, 1900.

22. Edwards, "Amer. Jour. of the Med. Sci.," April, 1888, p. 329.

23. Einhorn, "Arch. f. Verdauungs-Krankheiten," Bd. IV, H. 4, 1898.

24. Einhorn, Max, "Membranous Enteritis and its Treatment," "Medical Record," January 28, 1899, vol. IV, No. 4.

25 and 26. Ewald, "Membranous or Mucous Enteritis in Diseases of the Intestines," "Nineteenth Century Practice of Medicine," vol. IX, p. 265.

27. Ewald, "Deutsche med. Wochenschr.," 1893, No. 41.

28. Fernel, "Pathologia," livre VI.

29. Fielitz, "Berlin. klin. Wochenschr.," 1883, No. 46.

30. Findley, "Amer. Jour. of the Med. Sci.," 1875.

31. Fischl, "Prager Vierteljahresschr. f. Heilkunde," 1878.

32. Fischl, "Ein Beitrag z. Casuistic der nervösen Diarrhöen," "Prager Wochenschr.," 1891, No. 47.

33. Fitch, Wm. E., "Enterocolitis," "New York Medical Journal," August 18, 1900.

34. Fleiner, "Berlin. klin. Wochenschr.," 1893, No. 3 und 4.

35. Franke, "Mittheilungen aus den Grenzgebieten," u.s.w., Bd. I, 1896, S. 379.

36. Fruitnight, J. Henry, "Scorbutic Membranous Colitis," "Archives of Pediatrics," December, 1899.

37. Georg, Conrad, "Intestinal Sand," "Physician and Surgeon," Ann Arbor, Mich., May, 1900.

38. Glénard, "De l'Entéroptose," 1889.

39. Good, "The Study of Medicine," Cl. I, Ord. I, Species 7, vol. I, Philadelphia, 1825, p. 162.

40. Guyot, "Soc. Méd. des Hôpitaux," Bulletin, 1868.

41. Hall, J. N., "General and Local Infection by the Bacterium Coli," "Phil. Med. Jour.," December 30, 1899.

42. Henschen, "Wien. klin. Rundschau," 1896, No. 33.

43. Herzog, "The Dependence of Certain Neuroses and Psychoses upon Diseases of the Gastro-intestinal Tract," "Arch. f. Psychiatrie," Bd. XXXI.

44. Hoyer, "Ueber den Nachweis des Mucins in Geweben mittelst der Färbemethode," "Arch. f. mikroskop. Anatomie," 1890, Bd. XXXVI, S. 10.

45. Hughes, M. L., "An Acute Epidemic of Gastro-enteritis Attributed to Food-poisoning," "Lancet," November 4, 1899.

46. Isoard, "Contrib. à l'étude de l'entérite muco-membraneuse," Paris, 1883.

47. Jacusiel, "Berlin. klin. Wochenschr.," 1887, S. 253.

48. Jaksch, von, "Klinische Diagnostik," 1892, S. 195.

49. Jeannel, "Surgical Pathology of Duodenum (Pathologie chirurgicale du duodenum), "Archives Provinciales de Chirurgie," Paris, May 1, 1900.

50. Jurgeus, "Berlin. klin. Wochenschr.," 1892, S 357.

51. Kassowitz, "Beiträge z. Kinderheilkunde," Wien, 1892.

52. Kijanitzin, "Zur Frage nach der Ursache des Todes bei ausgedehnten Hautverbrennungen," "Virchow's Archiv," Bd. CXXXI, S. 436.

53. Kitagawa, "Beiträge zur Kenntniss der Enteritis membranacea," "Zeitschr. f. klin. Med.," 1890, Bd. XVIII, H. 1 u. 2.

54. Kobler, "Beitrag zur Kenntniss der Nierenerscheinungen bei acuten Darmaffectionen," u.s.w., "Wien. klin. Wochenschr.," 1890, No. 28–31.

55. Kolipinski, Louis, "Chronic Enteritis and Tubercular Enteritis Treated with Hypodermic Injections of Arsenic," "Medical News," August 11, 1900.

56. Koplik, Henry, "Nephritis Complicating Acute or Subacute Gastro-enteritis of Infants and Children: Its Symptomatology, Prognosis, and Treatment," "Medical Record," April 1, 1899, vol. LV, No. 13.

57. Krysinski, "Enteritis membranacea," Inaug. Dissert., Jena, 1884.

58. Laboulbène, "Recherches clin. et anat. sur les affections pseudo-membraneuses," 1861.

59. Langenhagen, de, "Semaine médic.," 1898, No. 1.

60. Lesage et Dauriac, "Des grands lavages de l'intestin grêle," "Gazette des Hôpitaux," October, 1893.

61. Letcheff, "De la colite muco-membraneuse chez les utérines," Paris, 1895.

62. Leube, "Spezielle Diagnostik der inneren Krankheiten," 1889, S. 256–292.

63. Leyden, von, "Deutsche med. Wochenschr.," 1882, No. 16 u. 17.

64. Leyden's, von, "Handbuch der Ernährungstherapie," 1898, Bd. II, 1. Abth., S. 309.

65. Litten, "Berlin. klin. Wochenschr.," 1888, No. 29.

66. Longuet, "Rec. de mêm. de méd. milit.," 1878, p. 467.

67. Löwenstein, "Deutsche med. Wochenschr.," 1889, No. 2.

68. Lyon, G., "L'entérite muco-membraneuse," "Gazette des Hôpitaux," 1889, No. 54.

69. Malsbury, J. O., "Acute Infantile Diarrhea," "Jour. Amer. Med. Assoc.," June 24, 1899, vol. XXIII, No. 25.

70. Marchand, "Berlin. klin. Wochenschr.," 1877.

71. Marshall-Hall, "Diseases and Derangements of the Nervous System," London, 1841, p. 153.

72. Mathews, "Disease of the Sigmoid Flexure," "The American Medical Quarterly," June, 1899.

73. Mathieu, "Soc. méd. des Hôpit.," 22. Mai, 1896.

74. Mathieu, "Traitement de l'entérite muco-membraneuse," "Gazette des Hôpitaux," 1894, No. 125.

75. Mathieu, "Therapeutique des maladies de l'intestin," Paris, 1895.

76. Mayor, "Quelques mots sur une varieté d'entérite iliaque," "Revue medecine de Suisse romande," 1893.

77. Mendelson, "Mucous Colitis, a Functional Neurosis," "Medical Record," January 30, 1897.

78. Newsholm, Arthur, "Epidemic Diarrhea," "Lancet," December 2, 1899.

79. Nonat, "Traité pratique des maladies de l'utérus et de ses annexes," 1860.

80. Noorden, von, "Ueber die Behandlung der Colica Mucosa," "Zeitschr. f. prakt. Aerzte," 1898, No. 1.

81. Nothnagel, "Beiträge z. Physiologie u. Pathologie des Darmes," Berlin, 1884.

82. Nothnagel, "Spec. Pathol. und Ther.," Bd. XVII, Theil 1, 1. Abth.

83. Nothnagel, "Darmkrankheiten," S. 139.

84. Ozenne, "Jour. de Medécine," December 31, 1893.

85. Pariser, "Deutsch. med. Wochenschr.," 1893, No. 41.

86. Plehn, F. W., "Inflammation of Duodenum and Resulting Reflexes," "Western Medical Review," December, 1899.

87. Potain, "Semaine Médic.," 1887, S. 341.

88. Quincke, "Enteritis from Protozoa (Ueber Protozoen Enteritis)," "Klin. Wochenschr.," November 13, 1899.

89. Richardière, "L'Entérite muco-membraneuse," "Union Méd.," 1895, No. 1.

90. Rosenheim, Theodor, "Pathologie und Therapie der Krankheiten des Darms," Wien und Leipzig, 1893.

91. Rosenheim, "Krankheiten des Darmes," Berlin, 1893.

92. Rosewater, N., "Enteroptosis Relative to Disorders of Digestive Tract," "Cleveland Journal of Medicine," Ohio, May, 1900.

93. Rossbach, in Nothnagel und Rossbach, "Handbuch der Arzneimittellehre."

94. Rothmann, M., "Deutsch. med. Wochenschr.," 1893, No. 41.

95. Rothmann, M., "Zeitschr. f. klin. Med.," 1893, Bd. XXII, H. 3 u. 4.

96. Rothmann, O., "Deutsch. med. Wochenschr.," 1887, No. 27.

97. Runeberg, "Von der diagnostischen Bedeutung des Eiweissgehaltes in pathologischen Trans- und Exsudaten," "Berlin. klin. Wochenschr.," 1897, No. 33.

98. Runeberg, "Klinische Studien über Transsudationsprocesse," "Deutsch. Arch. f. klin. Med.," Bd. XXXIV u. XXXV.

99. Saint Philippe, "Du Traitement de l'entero colite dysenteriforme des enfants, etc.," "Bulletin Medical," Paris, June 6, 1900.

100. Sasaki, "Virchow's Archiv," Bd. XCVI, S. 287.

101. Schmidt, "Experimental and Clinical Investigations upon the Tests of the Functions of the Intestines," "Deutsch. Arch. f. klin. Med.," December, 1898, Bd. LXI, H. 5 u. 6.

102. Schmidt, Ad., "Zeitschr. f. klin. Med.," Bd. XXXII, H. 3 u. 4, 1897.

103. Schmidt, Ad., "Verhandl. des Cong. f. innere Med.," 1895.

104. Schütz, "Bacteriologisch-Experimentelle Frage gastro-intestinaler Desinfektion," "Klin. Wochenschr.," Berlin, June 18, 1900.

105. Sée, Germain, "Des méprises entre les maladies de l'estomac et celles de l'intestin," "Bulletins de l'Acad. de Méd.," 1893, Décembre.

106. Sennertius und Morgagni, from J. J. Woodward, "The Medical and Surgical History of the War of the Rebellion," 1879, vol. I, p. 363.

107. Siredey, "Note pour servir à l'étude des concrétions muqueuses membraniformes l'intestin," No. 7–9, vol. I, "Union Med.," 1869.

108. Slifer, H. F., "Membranous Enteritis," "New York Medical Journal," September 22, 1900.

109. Stieler, "Ueber chronische Peritonitis," "Deutsch. Arch. f. klin. Med.," Bd. xvi, 1875.

110. Stieler, "Wien. med. Wochenschr.," 1880, No. 18 u. 19.

111. Strasburger.—Same reference as 102.

112. Thévenot, "Contrib. à l'étude du catarrhe int. à mucosité membraniforme," "Soc. méd. hep.," 1883.

113. Thomson, W. H., "Treatment of Membranous Colitis," "Medical News," New York, June 2, 1900.

114. Traube, "Die Symptome der Krankheiten des Respirations- und Circulationsapparates," Berlin, 1867.

115. Traube, "Zwei Fälle von Perforation-Peritonitis," "Gesammelte Beiträge," Bd. ii, S. 776, Berlin, 1871.

116. Traube, "Ueber den Einfluss starker und anhaltender Diarrhoen auf die Gestaltung peritonitischer Adhäsionen," "Gesammelte Beiträge zur Physiologie und Pathologie," Bd. iii, Berlin, 1878.

117. Traube, "Gesammelte Beiträge," u.s.w., Bd. ii, S. 1031, Berlin, 1871.

118. Troussard, "Enteralgia, Crises of, in Mucomembranous Enteritis (Les crises d'enteralgie au cours de l'enterocolite mucomembraneuse)," "Gazette Hebdomadaire," Paris, July 22, 1900.

119. Ullmann, "Deutsch. med. Wochenschr.," 1894, No. 2.

120. Vanni, "Rivista clinica," 1888, No. 4.

121. Van Swieten, "Diarrhea febrilis," tome I.

122. Virchow, "Ueber katarrhalische Geschwüre," "Berlin. klin. Wochenschr.," No. 8 u. 9.

123. Virchow, "Historisches, Kritisches, und Positives zur Lehre der Unterleibsaffectionen," "Virchow's Archiv," Bd. v, 1853.

124. Virchow, "Gesammelte Abhandlungen," Hamm, 1862.

125. Virchow, "Ueber den Gang der amyloiden Degeneration," "Virchow's Archiv," Bd. viii.

126. Virchow, "Neue Beobachtungen über amyloide Degeneration," "Virchow's Archiv," Bd. xi.

127. Wannebroucq, "Comptes rend. de l'Assoc. pour avanc. des sciences," Congrès de Lille, 1875.

128. Washbourne, J. W., "Gastro-enteritis, Dysentery, and Enteric Fever," "Brit. Med. Jour.," June 16, 1900.

129. Weber, L., "New Yorker medicinische Monatsschrift," 1892.

130. White and Golding Bird, "Clinical Society of London," 1896.

131. Whitehead, "Mucous Diseases," "Brit. Med. Jour.," 1871.

132. Woodward, "The Medical and Surgical History of the War of the Rebellion," vol. I, part 2.

133. Ziemssen, von, "Verhandl. d. VIII. Congresses f. innere Med.," Wiesbaden, 1889.

134. Ziemssen, "Künstliche Gasaufblähung des Dickdarms," "Deutsch. Arch. f. klin. Med.," Bd. xxxiii.

135. Ziemssen, "Deutsch. med. Wochenschr.," 1887, No. 35.

CHAPTER XX.

DYSENTERY.

Synonyms.—Colitis; German, *Ruhr* or *Dysenterie;* Latin, *Difficultas Intestinorum*. The term dysentery (*Δυσεντερία*) was first used by Herodotus in 484 B. C., who mentions that it was epidemic in the Persian army in Thessaly. The oldest reference to dysentery is that occurring in the papyros of Ebers; but Hippocrates, in 460 B. C., is the first one to have conceived and described dysentery as a substantive disease, his account of it being, on the whole, in agreement with the symptomatology as it is known to-day. He discourses on its occasional occurrence during autumn, its clinical course, and its complication with inflammation and abscess of the liver, and he knew that it led to ulceration of the intestines. Galen (Com. IV, in "Hippocrates de Articulis," liber XXXVIII, Ed. Kühn, XVIII, A. 724) asserts that Hippocrates included all kinds of bloody discharges from the bowels under the designation of dysentery. Galen described at length four varieties of bloody stools: (1) Those proceeding from plethora, as in the case of persons who have lost limbs or from the suppression of habitual hemorrhages; (2) those due to hepatic trouble—the hepatic flux, in which the stools resemble the washings of flesh; (3) those due to black bile—ex melancholia; (4) those due to ulceration of the intestine, which alone are properly to be called dysentery.*

Dysentery may be designated as a primary specific colitis, characterized by ulceration and sloughing of the mucous membrane, more or less persistent diarrhea, with rumbling, or borborygmi, the stools containing blood and mucus, and in severe cases consisting entirely of blood-stained mucus. Toward the end of the disease the evacuations may become putrefactive, containing sloughs and shreds of membrane. Clinically it is furthermore characterized by fever, abdominal pain, culminating in tenesmus and strangury. Anatomically the disease is characterized by catarrhal, pseudomembranous, and ulcerative processes in the colon. In many older writings the disease is divided into three types: (1) Endemic, which term is applied to dysentery existing always among a small number of persons or within a limited area, as contrasted with (2) epidemic, which is applied to the same disease when it involves large numbers of persons and is spread over a large area. (3) The third or sporadic form refers to the scattered and isolated cases.

---

* The complete history of this disease and that of the historical epidemics is given in the "Medical and Surgical History of the War of the Rebellion," vol. II, by J. J. Woodward. This is the most complete treatise on diarrhea and dysentery in existence, commanding the admiration of native and foreign clinicians, and an enduring monument to American clinical investigation.

Endemic dysentery is sufficiently characterized by its geographical distribution, occurring principally in the tropical and subtropical countries, by its clinical symptoms and the pathological picture, to justify its separation from other forms. The views concerning the etiology of this form are at the present day almost uniformly accordant, and it is believed to be caused by certain protozoa—the dysentery ameba, or the Amœba coli. There are tropical dysenteries, however, of which we know that they are not caused by the dysentery ameba. These are the endemic dysenteries that have been studied by the French clinicians Fournier and Antoine in Cochin China, certain of the epidemic forms of Japan, and the dysenteries that have been studied by my friend, Professor S. Flexner, among the American troops and the natives in the Philippines.

The epidemic forms occur with frequency, sometimes invading regiments or entire armies, causing more deaths than the weapons of the enemy. It sometimes spreads into a panepidemic, involving entire nations. With the exception of a type occurring in Japan, no specific cause of this form of the disease has so far been discovered.

The sporadic form of dysentery probably is represented by isolated cases of tropical dysentery or by types of the disease caused by mechanical or chemical injuries, foreign bodies, intestinal parasites, coprostasis, and unknown conditions. It is more correct to limit the conception of sporadic dysentery to those caused by mechanical and chemical irritants, since it has been recently demonstrated that a considerable number of cases classed under this type belong to the endemic or epidemic form. In the clinical sense of the terms various stages or degrees of dysenteric inflammation of the intestines have been designated as catarrhal, ulcerative, bilious, phlegmonous, gangrenous, etc.

Classification.

Principal forms: (A) endemic, (B) epidemic, and (C) sporadic.

A. Endemic or Tropical Dysenteries:
    (*a*) Endemic dysentery; (*b*) Cochin China dysentery; (*c*) the white flux, diarrhœa alba, pilgrim's diarrhea, sprue.

B. Epidemic Form:
    (*a*) Variety of epidemic dysenteries; (*b*) Japan dysenteries; (*c*) Philippine dysentery.

C. Sporadic Form:
    (*a*) Dysentery due to mechanical, chemical, catarrhal, or rheumatic origin; (*b*) dysenteries due to intestinal parasites—Bilharzia distoma, Trichocephalus dispar, and others; (*c*) dysenteries of unknown origin.

From a practical clinical standpoint the grouping into (1) tropical or amebic, (2) acute catarrhal, (3) diphtheric, and chronic dysentery is sufficient.

In a valuable contribution to our knowledge of idiopathic ulcerative colitis James F. Gemmel ("Idiopathic Ulcerative Colitis," London, 1898, p. 17) classifies this disease into two groups: (1) Idiopathic ulcerative colitis, a specific affection due to micro-organisms, beginning and throughout its course invariably limited to the colon. (2) Secondary ulcerative colitis, occurring in the course of specific diseases, as a more extensive manifestation of the disease of which it forms a part, not as a specific disease originating in or confined to the colon. The colon is in this manner implicated in typhoid fever, tuberculosis, syphilis, diphtheria, scarlet fever, and measles. The secondary ulcerative colitis Gemmel divides into specific and nonspecific. The specific types of the secondary variety have just been enumerated. The nonspecific types are those which I have classed under the sporadic forms caused by foreign bodies, chemical or mechanical irritation, etc.

Geographical Distribution.

There are few diseases which have exhibited so extensive a distribution in the history of the human race as dysentery. A. Hirsch

(" Handbuch d. historisch-geographisch Pathologie," Bd. II) asserts that dysentery had the widest distribution over the surface of the earth at all historic times, as it has in the present era, and that it spares no large domain of the inhabited portion of the earth. Ayres says of dysentery ("North American Medical Journal," 1860): "Where man is found, there some of its forms appear." Epidemic dysentery has occurred in this country, according to Noah Webster ("A Brief History of Epidemic and Pestilential Diseases"), as early as from 1749 to 1753; another epidemic occurred from 1773 to 1777; a third, from 1793 to 1798. During the last century numerous local epidemics occurred in all parts of this country. A full account of the history of these is given by J. J. Woodward (*l. c.*, p. 421). The volumes of vital statistics of the census report of 1870 and 1880 contain maps which illustrate the diversity in the geographical distribution of malarial fevers, diarrhea, and dysentery. It will be seen from the statistics and maps that neither diarrhea nor dysentery is most prevalent in those States in which malaria is most prevalent. There is more or less of dysentery in Baltimore every Summer, and in 1884 and 1886 I attended the inmates of Bay View Asylum through two epidemics of diphtheric dysentery, proved to be such by a large number of fatal cases upon whom the autopsies were made by Dr. Wm. T. Councilman. According to Woodward, acute and chronic dysentery was epidemic during the war of the secession in both armies. In the Union army he tabulated 259,071 cases of acute dysentery with 5576 deaths, and 28,451 cases of chronic dysentery with 3855 deaths. In addition to this he tabulates 1,269,027 cases of acute diarrhea with 4291 deaths, and 182,586 cases of chronic diarrhea with 30,836 deaths. The specimens collected at the Army Medical Museum and the autopsies recorded by Woodward show that the majority of cases of acute dysentery which proved fatal during our Civil War belonged to the diphtheric form. According to his statistics, the mortality of acute dysentery during the war was 1 death to every 57 cases among the white troops, and 1 to every 17 among the colored troops. Probably many of the cases tabulated as chronic diarrhea in his report would, if autopsies could have been made, have been arranged in the category of dysentery. According to Osler ("Practice of Medicine"), epidemics have occurred in Canada and Norway.

Etiology.

Effect of Climate and Temperature.—In climates lying in the equatorial zone endemic dysentery is prevalent at all times, especially during the hot months, becoming more rare during the cold periods. The disease is most intense during the late summer and beginning of autumn. In the Philippine Islands it has been observed that the climax of the frequency of endemic dysentery does not coincide with the period of highest temperature, but occurs at the end of the wet and the beginning of the dry season. This is in accordance with the statistics from other tropical countries. Great variations in the temperature and daily changes—for instance, cold and damp nights following very hot days—are believed to cause exacerbations of the disease.

Influence of Drinking-water.—Infected drinking-water is by far the most prolific cause of this disease. The infection in the endemic, as well as in the epidemic, form takes place through the drinking of impure water. Woodward, Gemmel, Kartulis, and many of the medical observers of endemics occurring among the British troops in tropical countries, corroborate this view. In 1887 I was a member of the corps of biologists studying at the Marine Laboratory of the Johns Hopkins University, located then at Beaufort, N. C. Shortly after the instructors and students had arrived at the laboratory an endemic of dysentery broke out. It was discovered by some of the investigators that the drinking-water, which was rain-water collected in a large cistern, contained many protozoa and bacteria; those who avoided its use or boiled the water were not taken with dysentery. Dr. T. Wesley Mills, of McGill University, Montreal, and myself, treated a number of our laboratory companions for dysentery. In the stools of two of these patients I found characteristic amebæ, to be described in the following.

The Influence of Diet and Putrefaction and Fermentation of Food.—The influence of these factors as causative agents in the production of dysentery is not so essentially different from the same rôle which they play in the etiology of diarrhea and enteritis as to require a detailed consideration here. To avoid repetition, the reader is referred to the influence of diet and unhealthy food in the chapters stated.

Influence of Occupation, Age, and Race.—Dysentery preferably attacks those classes that live under unfavorable hygienic conditions; common soldiers are more frequently attacked than officers. Poverty and misery are accompanied by conditions inviting the disease. No race is exempt from it; no age excepting that of the nursing infant is exempt from it; and there is no such thing as acclimatization.

Organic Excitants of Dysentery.—The most scientific investigations up to the present date have established the fact that dysentery in most of its forms is a parasitic disease, but that the different types are not due to the same parasite. Concerning endemic dysentery, there can be no doubt that it is due to the dysentery ameba. There can also be no doubt that certain forms of epidemic dysentery are due to micro-organisms.

Micro-organisms of Endemic Dysentery—the Dysentery Ameba, or Amœba Coli.—In 1859, long before the bacteriological era, Lambl discovered ameboid organisms in the evacuations of a child suffering from enteritis ("Berichte aus dem Franz Joseph Kinderspital in Prag," Th. I, 1860). In 1869 Basch described the occurrence of micro-organisms in endemic dysentery in Mexico ("Virchow's Archiv," Bd. XLV), but the first definite recognition of these microbes as protozoan organisms belonging to the class of amebæ was by Loesch, in St. Petersburg, in 1876. He discovered them in large numbers in a case of genuine dysentery ("Virchow's Archiv," Bd. LXV, 1875). The Amœba coli of Loesch varies in size from 0.02 to 0.03 mm. These amebæ are generally oblong in shape, have a somewhat coarsely granular protoplasm, which, however, is hyaline in the pseudopodia. The limiting membrane is not distinct. Within the body of this ameba is a pale, spherical nucleus and several vacuoles, most of which are digestive vacuoles. One is the so-called contractile vacuole, but it is almost impossible to determine which is the special contractile vacuole, as they all change their size, becoming smaller or larger, during the movement of the ameba assuming an elongated or irregular shape. The protoplasm frequently contains foreign matter, such as starch granules, blood-corpuscles, micrococci, vibrios, and bacilli. According to Loesch, the organism moved in the following manner. At any part of the surface of its body a rounded, transparent protrusion is formed, which may soon be again retracted, or, rapidly increasing in size, be extended more and more outward. Eventually this may form a finger-shaped projection or pseudopodium. The entire pseudopodium may be retracted, or the granular protoplasm may pour out into it. Several pseudopodia may be extended simultaneously, whereby the general contour of the parasite is changed from round to oval or to quite an irregular shape. The rate of progress is very slow. In one minute it barely moves the distance of its own body, though this varies greatly. In some specimens of dysenteric stools examined by myself on a warm stage and with the micrometer I observed specimens which moved through a distance of from 80 to 120 μ in one minute. The parasite is best examined in fresh stools immediately after evacuation. In the bloody stools of endemic dysentery they are, as a rule, present in masses. They do not stain very well with anilin colors, but take up eosin readily. According to Lutz ("Centralbl. f. Bakteriol. u. Parasitenk.," Bd. x, S. 243), the stools contain Charcot's crystals in addition to the amebæ. The most practical stain for Amœba coli is that suggested by F. B. Mallory, in the "Journal of Experimental Medicine," volume II, page 529.

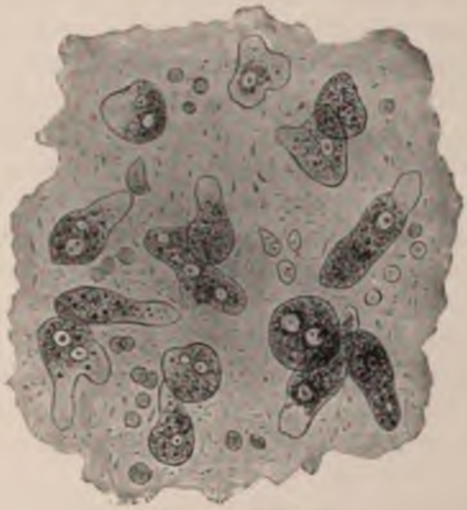


FIG. 22.—AMŒBA COLI.

After Lambl's observation, Lewis in 1870, and Cunningham in

1871, found ameboid organisms in the dejections of cholera patients in India; Normand found amebæ in 1879 in two cases of entero-colitis; Grassi discovered amebæ in dysentery, typhoid fever, cholera, pellagra patients, as well as in healthy persons. Perroncinto in 1882 found these parasites in a case of enteritis. This, according to Kartulis (*l. c.*), was the state of the history of investigations on the Amœba coli up to the year 1883, when R. Koch demonstrated amebæ in sections of intestines of individuals that had died of dysentery in India and Egypt. He was of the opinion that the amebæ were in some etiological relation to the disease. Soon thereafter Kartulis observed the amebæ in 500 cases of endemic Egyptian dysentery. He found them absent in cases of all other diseases examined by him. The presence of the parasites was demonstrated in sections of intestines from cases of endemic dysentery, as well as in the hepatic abscesses following this form of dysentery. Hlava confirmed these studies by finding the amebæ in sixty cases of endemic dysentery in Prag.

The contributions of our American countrymen to this interesting subject have been numerous and important, and will be found in the tabulated literature at the end of this chapter. J. J. Woodward (*l. c.*, p. 371), although aware of the discoveries of Lambl, Loesch, and Lewis relating to the Amœba coli, seems to have made no personal observation concerning this parasite in the enormous clinical material which either he personally or his associates had under their care.*

The following are some of the Americans who have published observations on the Amœba coli: Councilman ("Boston Med. and Surg. Jour.," 1892), Councilman and Lafleur ("Amebic Dysentery," "The Johns Hopkins Hospital Reports," 1891), Osler ("Centralbl.

---

* During the Civil War J. H. Salisbury, in his report to the Surgeon General of the State of Ohio, Columbus, 1865, on the chronic diarrhea of our soldiers, described, under the name of algoid cells, certain forms which he observed in the stools of refugees and which he compared to Protococcus cocoma. This writer embraced all the various forms of dysentery which occurred among the troops during the war under the name of chronic diarrhea. He describes jelly-like lumps, small masses of living gelatinous matter, holding that they are made up of large algoid cells and filaments enveloped in a jelly-like substance. He also described large numbers of these cells, some of them larger than the species torula. J. J. Woodward, in quoting this author, does not, however, seem favorably impressed with the trustworthiness of his observations.

f. Bakteriol. u. Parasitenk.," Bd. VII, S. 736), Dock, Musser, Alfred Stengel, Wm. T. Howard, Jr. ("Medical News," 92), Eichberg, Simon, and Lutz. I am particularly familiar with the researches of Councilman and Lafleur. Through kindness of the former I was enabled to study the Amœba coli, which he discovered in fourteen out of fifteen cases of endemic dysentery. In the fifteenth case the amebæ were found at the autopsy. These observers came to the conclusion that the Amœba coli was the exclusive cause of endemic dysentery, and described histological peculiarities by which this form differs from other types of dysentery. The name amebic dysentery is original with them, and their investigations, as well as those of the other Americans, give sufficient evidence that this disease is not so rare in our country as was formerly assumed. The theory of the amebic causation of endemic dysentery has been contested by a number of investigators, among whom are Grassi, Cunningham, Casagrandi, Barbagallo-Arapisardi, and Gossi. A number of these observers have found these parasites in other intestinal affections, such as chronic enteritis, chronic proctitis, typhoid fever, cholera, etc. Cunningham, Grassi, and Calandruccio have called attention to the fact that amebæ can be found in the stools of healthy individuals. Schuberg found them in ten of twenty sound individuals to whom Carlsbad salts had been given as a laxative. In the same individuals the amebæ were not found when the stools were solid. The amebæ either withdraw in consequence of physical and chemical conditions existing in the fecal matter of the intestines, or they are destroyed by the acid fermentation. Castor oil exerted a deleterious effect on the amebæ in Schuberg's experiments. It is conceivable that there may be several different types of amebæ in the human intestines, of which only one may be pathogenic. This has been established for cats by Quinke and Roos, who found that, in addition to the pathogenic dysentery amebæ, the feline intestine contains another species—Amœba coli mitis—which is not pathogenic for cats. A number of the observers quoted fail to recognize any difference between the amebæ occurring normally in the intestines, and those occurring in dysentery. Celli and Fiocca base their opinion that the Amœba coli is not the cause of dysentery upon experiments made upon cats. They claim to have produced dysentery by injecting material in which the amebæ were destroyed either by distilled water or by warming the infected material. They also consider that the experiment of Kruse and Pasquale, who produced dysentery in cats by infecting them with pus from a hepatic abscess containing amebæ without any admixture of bacteria, is not flawless, because the pus had been cultivated only on agar. According to Celli and Fiocca, that kind of pus, when grown on bouillon, is still shown to contain bacteria. Kartulis, however, asserts that this hepatic abscess pus proved to be sterile not only when he inoculated it on agar, but also when it was inoculated on bouillon. Celli and Fiocca base their opinion that the Amœba coli is not pathogenic principally upon four assumptions: (1) Because there are forms of dysentery without amebæ. (2) Because it is possible to produce a dysentery in cats which will show no amebæ in the discharges or colon—by inoculating them with dysentery stools or cultures which contain amebæ and bacteria. In a similar way dysentery can be caused in cats after the amebæ have been killed by heat and only bacteria and their toxins are inoculated into the animal. (3) Because the amebæ are widely disseminated. In Egypt the Amœbæ coli are very frequent; therefore their frequency in dysentery is natural, and they are in an analogous manner found in healthy individuals who are not afflicted with dysentery and never had it. (4) In the intestines of patients afflicted with other diseases and even in normal individuals different species of amebæ are found.

Kartulis asserts that he has investigated these objections and repeated all the experiments of Celli and Fiocca in all their details without being able to confirm them. While he admits that amebæ occur in healthy individuals, he holds that they are entirely different from the dysentery amebæ, and can perhaps be looked upon as innocent intestinal parasites. In many hundreds of cases of normal individuals Kartulis found the amebæ only three times. All the opponents of the amebic theory of dysentery, with the exception of Celli, studied the subject in their own countries, and not in the tropics. Nothing is said in their reports of autopsies or the condition of the amebæ in the ulcers of the colon.

The controversy suffers somewhat from the fact that up to the present time it has been impossible to cultivate amebæ in a manner that will meet all the demands from a purely technical standpoint. Casper O. Miller ("The Cultivation of Amebæ," "Contributions to the Science of Medicine," dedicated to William H. Welch by his pupils, Baltimore, 1900, p. 211) has not succeeded in cultivating amebæ without the presence of bacteria. The experiments of Kruse and Pasquale, which were confirmed by Kartulis, carry great weight, however, because they produced dysentery in cats by inoculating them with amebæ from dysenteric liver abscesses which were free from bacteria. Kartulis (*l. c.*, p. 28) sums up the proofs of the pathogenic character of the dysentery amebæ in the following manner: (1) The constant occurrence of the amebæ in every case of typical endemic dysentery, with the exception of the dysenteries occurring in Cochin China. (2) The occurrence of the amebæ in the walls of dysenteric ulcers; their absence in the intestinal ulcers of different origin. (3) Inoculation of cats with feces of amebic dysentery will cause the disease in these animals. (4) Inoculation of cats with pus from liver abscess containing amebæ but no other organisms will produce dysentery. (5) Inoculation of other microbes occurring in dysenteric stools will not produce dysentery in animals—the experiments of Celli and Fiocca excepted. (6) Inoculation with amebæ found in healthy human beings will not produce dysentery in cats.

Causes of Other Forms of Endemic Dysentery.—In some publications on the subject there seems to be a tendency to classify dysentery into varieties or types according to the micro-organisms which have been successfully isolated and cultivated in pure culture. According to M. de Silvestri ("Compt. rend. Soc. de biol.," Fevrier, 1895), there exist several varieties of dysentery due to different micro-organisms. One variety is attributed to a diplococcus; another to a bacillus; and another to the Amœba coli. Dr. J. H. Goodliffe, who worked up the bacteriological side of the publication on "Idiopathic Ulcerative Colitis" by James F. Gemmel, has isolated an organism which is always present in undoubted cases of this disease, and is capable of cultivation outside of the body on artificial media, thus conforming with two out of the three fundamental conditions necessary to establish its claim as the specific cause of this disease. The third condition, the reproduction of the same disease by inoculating the cultivated bacillus, he has, unfortunately, not been able to verify (*l. c.*, p. 124). Goodliffe's bacillus closely resembles the colon bacillus—so much so that, in his earlier investigations, he was inclined to believe that it would prove to be one of the many varieties of this organism, which would have been in accordance with the view of Arnaud ("Annales de l'Institut Pasteur," 1894, No. 7) and others, who hold that dysentery is due to a pathogenic variety of Bacillus coli, of which many varieties have been described, all having morphological resemblances but differing in some reaction or mode of growth. Normand, in studying the dysentery of Cochin China, found amebæ in the discharges at first, but later on attributed it to the Anguillula stercoralis; other observers, however, have found this parasite in other diseases, and even in healthy individuals in Cochin China. Calmette, in studying the dysenteries in Cochin China, comes to the conclusion that it is caused by the Bacillus pyocyaneus in association with a streptococcus. Chantemesse and Widal isolated from the discharges of tropical dysentery a bacillus with rounded edges that stained poorly with anilin dyes; on intraperitoneal injections into guinea-pigs it caused peritonitis, pericarditis, and in one case intestinal ulceration.

Causes of Epidemic Dysentery.—The views of various investigators differ widely concerning the micro-organisms which cause epidemic dysentery. There can be no doubt that there must be different types of epidemic dysentery caused by different micro-organisms. It seems probable that S. Flexner has discovered an organism which is the real cause of the dysentery in the Philippine Islands, and that Shiga and Ogata have found the cause of the epidemic dysenteries in Japan. Celli and Fiocca have advanced a complicated theory, according to which the Bacillus coli communis, when associated with a typhoid-like bacillus, more rarely with a streptococcus, is converted into the Bacillus coli dysenteriæ. Other observers (Maggiora and Laveran) assert that the Bacillus coli alone can cause dysentery. Kartulis has tested the animal inoculations of Celli and Fiocca, and has been unable to produce dysentery in cats by inoculating them with the colon bacillus cultivated according to the method of these observers.

In determining critically the value of the bacteriological evidence in explanation of the etiology of dysentery, one can not fail to be impressed with the necessity of a master mind for reinvestigating the various experimental results and classifying them according to their merits, and bringing to light the defects in the technic that may explain the differences of various investigators. Gemmel (*l. c.*) asks very pertinently: "Wherein do the varieties of dysentery said to depend on the colon bacillus, the Amœba coli, a diplococcus, and the various bacilli of other investigators, differ from each other, except in the fact that the ultimate issue of cultivation experiments results, in the hands of different observers, in the survival of different micro-organisms?" Further investigation will show, no doubt, that many of the organisms isolated as the cause of dysentery by different observers are identical, and the slight cultural, tinctorial, or metabolic differences due only to different methods employed or to errors in technic.

An endemic of dysentery which occurred at Hartwick, a small hamlet in Otsego County, New York, was traced by Dr. A. J. Lartigau to the Bacillus pyocyaneus, for in fifteen cases of this dysentery this organism was present in all of them and also in the drinking-water ("Epidemic Dysentery caused by Bacillus Pyocyaneus," August J. Lartigau, "Jour. of Exper. Med.," vol. III, p. 595). Calmette ascertained the cause of endemic enterocolitis of Cochin China to be this same Bacillus pyocyaneus, which was also found to be the causative agent by Adami in an epidemic among children in Canada.

Pathological Histology.

It is not feasible nor practicable to go into the history of the pathology of dysentery. Suffice it to state that the American contributions of note begin with those of Woodward (*l. c.*, p. 563). The photographic and lithographic plates and the histological illustrations in this exhaustive work are unsurpassable. Woodward describes cases of genuine epidemic dysentery under the name of "catarrhal and diphtheric inflammations of the mucosa of the colon, eventuating in suppurative follicular ulceration." He also describes and pictures the pathological histology of endemic types of dysentery, showing marked thickening of the intestinal walls and cavities in the submucosa communicating with the intestinal lumen by small openings. Another important contribution to this subject is that by the author's teacher, Professor Wm. T. Councilman, which was published by him, together with Dr. Lafleur. These authors investigated the collection of the intestines in the Army Medical Museum at Washington brought together by J. J. Woodward, and confirmed his descriptions. Councilman and Lafleur investigated the pathological histology of the epidemic as well as the endemic dysentery, or, as they called it, the diphtheric colitis and amebic dysentery.

1. **Endemic Forms.**—According to Councilman and Lafleur, the amebic dysentery is identical with the follicular dysentery of other authors; still they do not look upon the solitary follicle as the point of origin of the ulceration, which is the principal point of difference between their description and that given by Kruse and Pasquale ("Zeitschr. f. Hygiene," 1893, Bd. XVI), who attribute to the solitary follicle the main rôle in the suppurative inflammation. Kartulis (*l. c.*, p. 53) agrees with Kruse and Pasquale in assigning the origin of the suppurative inflammation not only to the submucosa, but also to the solitary follicles, but admits that the latter involvement is much rarer than that of the submucosa. In ulcerations that have far advanced the origin of the disease can no longer be recognized, but in those that have not yet fully developed one is sometimes still able to observe the beginning ulceration in a follicle. In many cases, however, it is quite evident that the ulceration begins in the submucosa, while adjacent follicles are quite intact. According to Councilman and Lafleur, the characteristics of this form are the invasion and alteration of the submucosa, but a large variety of alterations, inflammations, and ulcerations were observed in different cases, and even in one and the same case at different localities of the colon. The increase in thickness of the intestine is mainly found in the submucosa. Small, projecting, knob-like indurations are described, in which minute cavities occur that are filled with a gelatinous pus; these cavities frequently communicate by means of submucous tunnels, and vary in size. The openings by which they communicate with the intestinal lumen are occasionally no larger than the head of a pin; sometimes, however, as large as the cavity itself. The surrounding bordering mucosa is deeply undermined by submucous fistulous passages. The ulcers are generally filled with the same material as the cavities. Microscopically examined in a fresh state, this material was found to consist of amebæ, large,

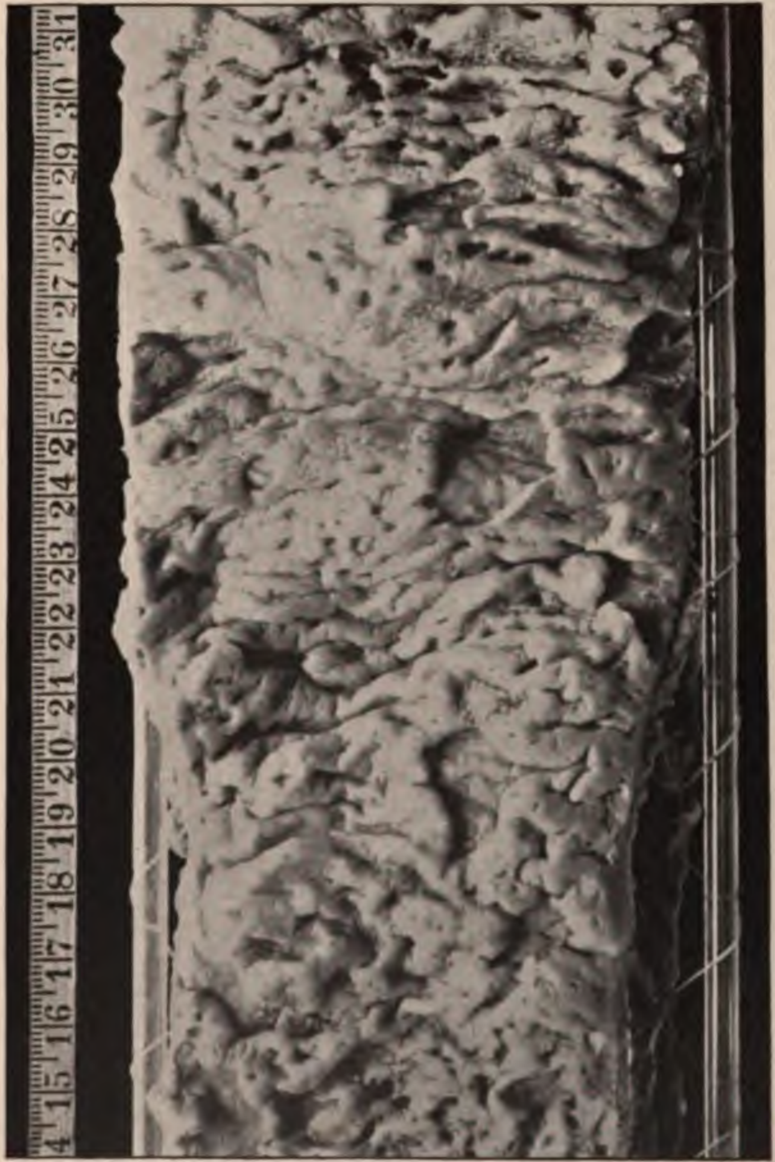


FIG. 23.—AMEBIC DYSENTERY (COLON).

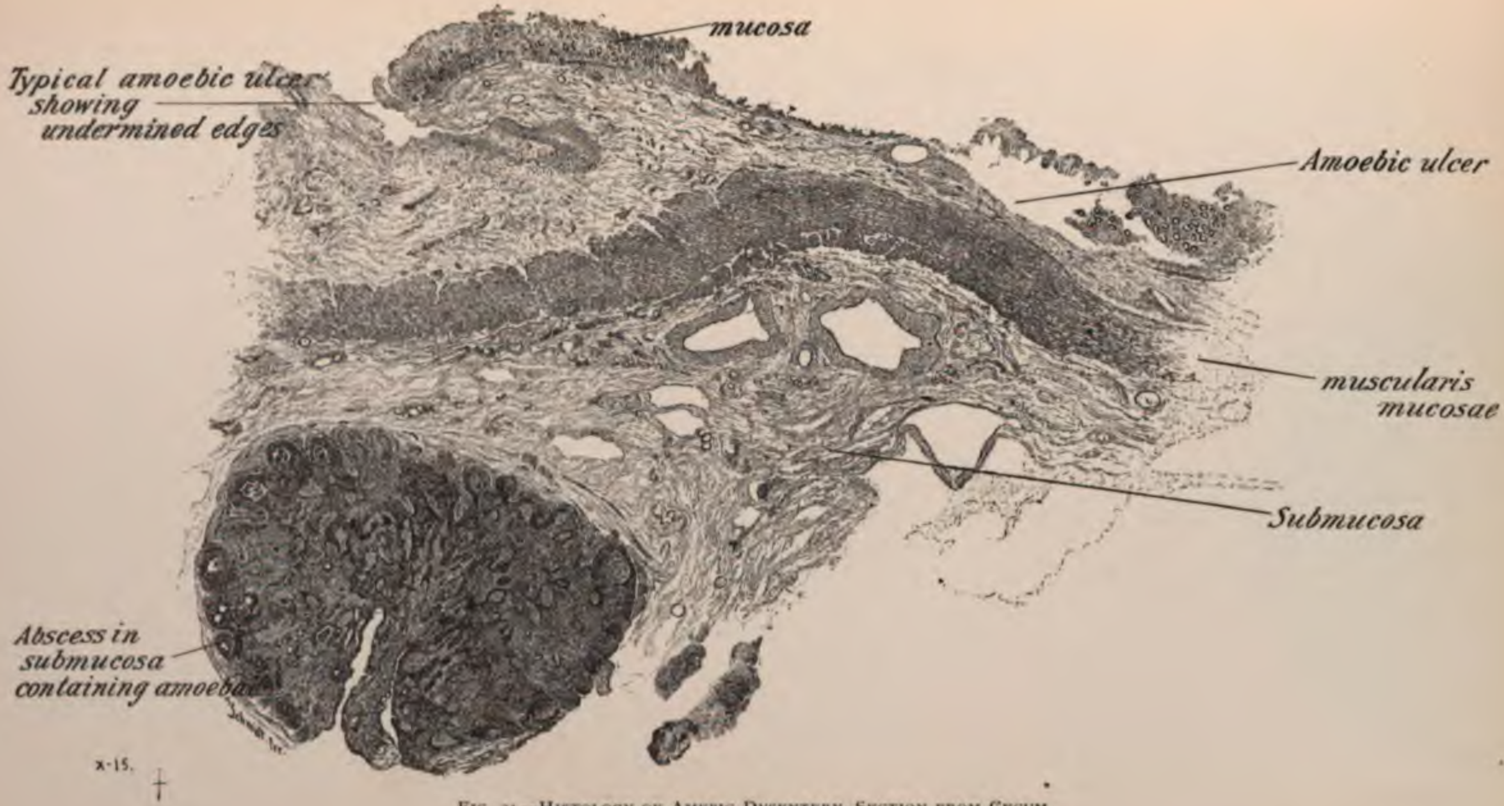

mucosa
Typical amoebic ulcer showing undermined edges
Amoebic ulcer
muscularis mucosae
Submucosa
Abscess in submucosa containing amoebae
x-15.

FIG. 24.—HISTOLOGY OF AMEBIC DYSENTERY, SECTION FROM CECUM.

round, and swollen cells, red blood-corpuscles, and pus-cells. The process seems to extend by progressive infiltration and softening of the submucosa, with subsequent necrosis of the superjacent mucosa. Generally the mucosa itself is least involved. Councilman and Lafleur distinguish four forms of ulcers in amebic dysentery: (*a*) Those with purulent infiltrations, softening, and cavities in the submucosa; small openings through the mucosa lead into these cavities, which communicate with neighboring ulcerations by submucous, fistulous passages. (*b*) Ulcers resembling simple excavations of the thickened submucosa, the edges of which are slightly undermined. (*c*) Ulcers presenting smooth edges and a clean base. (*d*) Ulcers covered with incrustations.

The process begins and has its foundation in a diffuse hemorrhagic catarrh of the colon, and it is probable that, in slight cases, it does not go beyond this. The typical ulcer of endemic dysentery is characterized also by a very small secretion or purulent exudate. The suppuration, if it occurs at all, is localized in certain places on the surface of the mucosa. The foci in which the amebæ are embedded are generally free from purulent infiltration. There are slight deposits of fibrin, occurring disseminated in the necrotic tissue, but they are not frequent. By this fact the amebic dysentery is distinguished from the diphtheric form.

Councilman and Lafleur assume that typical ulcers of amebic dysentery are caused by the amebæ alone, and all other lesions are the result of the cooperation of bacteria. Kruse and Pasquale assert that the amebæ and the bacteria always work together in producing the pathological changes of the intestinal wall. It can not be definitely stated, however, how and in what manner the amebæ penetrate into the intestinal tissues. According to Kovacs, the amebæ enter only such places of the mucosa where a previous lesion of continuity in the tissue existed. According to Roos, the cells seem to die or are destroyed under the influence of a toxin secreted by the amebæ. Concerning the question whether pathogenic bacteria cooperate with the amebæ in producing the ulcers, it should be stated that Kartulis (*l. c.*, p. 53) has not been able to demonstrate bacteria in the deeper layers of the intestinal walls in recent ulcerations. The bacteria were usually restricted to the mucosa or edges of the ulcer. In rare cases of ulcers complicated with other processes the bacteria accompanied the amebæ into the depths of the tissue. According to this author, the beginning of the process is either a destruction of the epithelium or an invasion of the solitary follicle; then the amebæ penetrate along the interstices, lymphatic spaces of the tubular gland, and provoke an intense inflammation, which is characterized by engorgement of the vessels and ecchymoses and swelling of the glandular cells, eventuating in necrosis. The further advance of the parasites necessitates breaking through the muscularis mucosæ, which has probably been softened by an inflammatory change and infiltration with leukocytes. The appearance of some specimens led Kartulis to believe that the amebæ do not break through the muscularis mucosæ into the submucosa, but reach the latter by passing through the lymph-channels of the muscularis mucosæ. The further developments are an extensive suppurative process under the influence of which the submucosa seems gradually to melt away. There is an active proliferation of round cells in the mucosa, extending also toward the lumen of the bowel, by which time an opening has been made toward this lumen through a softening of the muscularis mucosæ and progressive destruction of the gland tubules.

The large round cells, which are described by Kartulis (*l. c.*, p. 56) as occurring in the deeper layers of the submucosa in amebic dysentery, and claimed not to be found in any other ulcerative processes, have been described and pictured by Woodward (*l. c.*, pp. 468 and 469).

There are three secondary processes which accompany the ulcerative type of amebic dysentery. These are: (*a*) The fibrinous exudation; (*b*) the diphtheric or croupous exudation; (*c*) the hyperplasia of the intestinal mucosa.

(*a*) The fibrinous exudation is a result of croupous or diphtheric inflammation, which is characterized by deposits of fibrinous strata over the glands of the mucosa. This has also been described by Woodward. A stratum two or three millimeters in thickness—a small round-cell infiltration—lies immediately on the mucosa, and extends down into the spaces between the tubular glands: in some cases down into the submucosa. The glands of the mucosa are forced asunder and isolated by this infiltration. Epithelial cells are rarely present, and if so, they no longer stain. The submucosa is enormously infiltrated and edematous. The most frequent micro-organisms are the streptococci, which are found in the submucosa, as well as on the surface in the croupous deposits. (*b*) The diphtheric inflammation is a necrosis of the mucosa subsequent to exudation. The epithelium of the glands gradually disintegrates, swells up, loses its affinity for stains, and is gradually converted into an amorphous necrotic mass. Under the necrotic zone more or less fibrinous exudation takes place, which may extend into the submucosa. When complete necrosis of the mucosa has taken place, the Weigert stain reveals nothing but a few strands of fibrin and numerous colonies of bacteria. (*c*) The third exudative process has been described by Kartulis under the name of fibrinous hyperplasia of the mucosa. The process is brought about by fibrinous exudation on the surface of the mucosa and in the interstices of the gland tubules. Numerous amebæ and colonies of micrococci and of short small bacilli occur in the exudate. The vessels of the submucosa the walls of which have undergone marked hypertrophy contain amebæ and short bacilli. The diseased intestinal wall may acquire an enormous thickness—in places 225 mm. In one specimen of amebic colitis which I measured personally the thickness amounted to 232 mm.

The peritoneum is often involved in the process when the ulcerations extend to the serosa. Local peritonitis or adhesion of the intestinal serosa to the parietal peritoneum or that of other organs has been described. Kartulis has observed adhesions which were so firm that the suppurative amebic process extended through the abdominal muscles and discharged outwardly. Amebic involvement of the mesenteric glands has not been observed.

2. **Pathological Histology of the Epidemic Form.**—Epidemic dysentery has two principal stages: (*a*) The catarrhal and (*b*) the diphtheric or croupous. The catarrhal stage is essentially the same as that occurring in an intense acute catarrhal colitis. The mucosa of the colon is injected, and numerous ecchymoses are seen, especially on the highest portion of the folds. The mucosa seems to be striated by dark-red stripes. This condition is beautifully shown in the illustration of Woodward (*l. c.*), and also of J. F. Gemmel (*l. c.*). The mucosa is extensively destroyed by ulcerations; the entire wall of the colon is sodden, infiltrated, and covered

by a layer of either grayish or grayish-red mucus, beneath which the inflamed and ulcerated mucosa can be distinguished; or, in other cases, the mucosa is covered by the diphtheric or croupous exudation. In some cases of advanced dysentery the mucosa is covered with a furfuraceous or bran-like layer, which is composed of a mixture of necrotic epithelium and mucus, frequently mixed with blood and pus-cells. In many localities the mucosa is entirely detached and presents a condition similar to that described by Wm. B. Booker in the intense gastro-enteritis of children ("Johns Hopkins Hospital Report," vol. VI). Histologically the mucosa as well as the submucosa present numerous hemorrhagic foci; the epithelium of the tubular glands is swollen and clotted, and in the submucosa numerous extensive accumulations of round cells are observed. J. F. Gemmel (*l. c.*) devotes almost the entire paragraph on microscopical appearances to the question whether the surface of the mucous membrane is first attacked or the solitary follicles in idiopathic ulcerative colitis, and maintains that the involvement of the solitary follicles is always secondary to that of the mucosa.

As the process described above advances, the infiltration extends into the follicles, and the so-called follicular ulcerations and suppurations ensue, which may lead even to gradual destruction of the muscularis mucosæ and the mucosa. Detachment or sequestration of the mucosa in circumscribed areas leaves behind small necrotic islands which, if the case should go on to healing, are recognizable as flat, pigmented scars.

The croupous or diphtheric exudation is brought about by three conditions: (*a*) Round-cell infiltration with (*b*) contemporaneous necrosis of the epithelium of the mucosa and (*c*) appearance of fibrinous exudation in the submucosa. The process first develops on the summit of the involutions and folds, and as it progresses the mucosa eventually becomes gangrenous. The ulcers, which are flat and correspond to the configuration of detached portions of the necrotic mucosa, have their bases in the muscularis mucosæ; exceptionally, the base of the ulcer may be found in the submucosa or the true muscularis.

Japanese Dysentery and the Dysentery of the Philippine Islands.—The pathological anatomy of this form of dysentery is not so well known as that of the forms hitherto described, because autopsies are obtained with great difficulty in Japan, and the number of dysentery cases which have been examined postmortem is not large. In the January number of the "Public Health Reports," published by the Treasury Department of the United States Marine Hospital Service, volume xv, No. 1, January 5, 1900, Dr. Stewart Eldridge, U. S. M. H. S., Sanitary Inspector at Yokohama, gives an interesting account of epidemic dysentery in the past twenty years in Japan. From the pathological portion it is evident that the destruction of the tissues of the colon appears to progress from the surface downward, or to be the effect of general interstitial necrosis from croupous infiltration. The summits of the folds of the large intestine, or those of the valvulæ conniventes of the small intestine, when this is involved, are generally attacked first. Perforation is unusual. This agrees also with the observations of Woodward, Gemmel, and my own clinical experience. The characteristic undermined ulcers and the follicular, pustule-like ulcer communicating with other similar pus-cavities by submucous tunnels, so graphically described by Councilman and Lafleur, and the extensive destruction of the submucosa are seldom met with in this form of dysentery. The mesenteric glands are frequently enlarged and hardened, or, if life is sufficiently prolonged, they may suppurate and become caseous. As I have stated above, this is very rarely observed in amebic dysentery. Dr. Kiyoschi Shiga, assistant to Professor Kitasato, in charge of the laboratory of the Tokio Institute for the study of infectious diseases, has published the results of his studies of dysentery ("Saikin Gakku Zasshi," Tokio, December, 1897; also "Centralbl. f. Bakteriol.," Bd. xxiv, No. 22, 23, and 24, 1898). Dr. Shiga concludes that he has found the specific cause of dysentery at present ravaging Japan, and has called it the Bacillus dysentericus. He has produced an antitoxic serum for the treatment of this dysentery, to which I shall revert in the clinical portion of this article. Dr. S. Flexner, in his studies of the dysenteries occurring among our troops in the Philippines, has also isolated an organism from the stools which he believes to be the specific cause of this disease.

Bacillus Dysentericus Shiga-Flexner.—The following is taken from a personal letter from Dr. Flexner concerning this organism. The bacillus which he has isolated from the cases of Philippine dysentery differs from all the bacilli hitherto described excepting the one isolated by Shiga. The organism is widely different from the Bacillus coli communis and approaches much more nearly the Bacillus typhosus in its properties. It is a slightly motile bacillus that stains by the ordinary methods but not by Gram's method. It grows upon ordinary culture-media and in a manner very similar to the Bacillus typhosus. It is incapable of fermenting any of the sugars with the production of gas. In size it approaches the colon typhoid group. It is pathogenic for animals such as the mouse, rabbit, guinea-pig, cat, dog, etc., and in the cat and dog produces intestinal disturbances associated with necrosis of the mucous membrane and even ulceration. It gives specific agglutinative reaction with the blood of dysenteric cases, but not with normal blood. It has been found in a considerable number of cases of dysentery, and is absent from the normal intestine as well as from the intestine in other diseases. It is most abundant in the early stage of dysentery, and grows progressively less easily demonstrable as the cases pass into the chronic type. It may also appear in the mesenteric glands and in the liver, and usually does not give rise to a general infection of the body.

4. Pathological Histology of Sporadic Forms.—The sporadic forms of dysentery that have come under my observation in this country are either isolated cases of amebic dysentery or belong to the epidemic ulcerative colitis. This agrees with the observations of Wm. T. Howard, Jr., Dock, Osler, Thayer, Flexner, Councilman, Lafleur, and other American investigators. The pathologico-anatomical picture can therefore not be sufficiently distinct from the types already described to justify a separate description. Probably the dysenteries that occur as a result of irritation by foreign bodies, coproliths, or intestinal parasites might be classed as genuine sporadic forms.

To this category belongs also the so-called *Bilharzia dysentery*, which is an inflammation of the colon caused by irritation through deposits of the egg of Distoma hæmatobium. This trematode is, according to Bilharz, found in at least 50 per cent. of the lower classes in Egypt. It lives in the venous system, particularly in the portal vein and the veins of the spleen, bladder, kidneys, and mesentery. When it invades the colon, the eggs are found in the submucosa; rarely, in the mucosa. It generally causes small polypoid excrescences. The ulcers are seated only in the mucosa. The entire intestinal wall may be hypertrophied in all its layers, but particularly in the submucosa by deposits of the eggs of this parasite. Hematuria is a first and most constant symptom, but in some cases the symptoms are very similar to those of ulcerative dysentery.

Symptomatology.

Prodromal Stage.—So far as I could determine from the anamnesis and personal accounts of patients of sufficient intelligence to attribute importance to their edicts, the prodromal symptoms of dysentery that are most often noticed are a feeling of pressure, distention, fullness, and weight in the lower abdomen, together with headache, anorexia, and general indisposition. Personally I have had two attacks of dysentery and can confirm these statements, adding that in the small epidemic observed at the Johns Hopkins Marine Laboratory at Beaufort in 1886 a number of the men complained of the symptoms mentioned before there was any diarrhea. A number of patients who have hitherto suffered with pronounced constipation suddenly discover that their bowels move regularly, but after four or five days this regularity is converted into a painless diarrhea. Usually the patients are not presented for examination until the diarrhea is established. In regions where the water is known to be unhealthy—in asylums and institutions for the insane where endemics of dysentery have existed before—this diarrhea should be looked upon as prospective dysentery and precautions taken accordingly. I have known this diarrhea to develop and eventuate into dysentery where I had every reason to believe that no error in diet had been committed. The patients had simply taken in so much of infected drinking-water that no matter how careful the diet, the infection took place nevertheless. As a general rule, however, there is a history of some indiscretion of diet.

There are cases which have no prodromal symptoms at all, but which set in at once with bloody and mucous stools evacuated under great pain and tenesmus. In the beginning the general condition of the patient in dysentery is hardly affected. Dysenteries of intense severity begin at once with chills, fever, nausea, vomiting, anorexia, and great depression. There is no typical or classical clinical picture of dysentery. It may vary greatly in its symptoms, according to the character of the disease, its seat, and its extent. The differential diagnosis between the various types of dysentery can generally be ascertained by investigation into the etiology, time, season, and geography or topography of its origin, and clinically from the progress of the disease and the macroscopic and microscopic appearances of the evacuation. The beginning of dysentery presents, as a rule, the clinical symptoms of an acute enteritis; with this difference, however, that all forms of dysentery exhibit varying stages, which I have already described as the catarrhal, ulcerative, and diphtheric stages. In the endemic dysenteries, those probably caused by bacteria (bacilli of Shiga, Ogata, or Flexner) as well as those caused by the Amœba dysenteriæ show mainly an ulcerative type, while the catarrhal varieties, with the diphtheric croupous exudations, prevail in the epidemic types. The duration of epidemic types is, as a rule, short, though varying considerably in the various epidemics; endemic dysentery has a tendency to become chronic.

Endemic Type—Tropical or Amebic Dysentery.—These types have a catarrhal and an inflammatory or ulcerative stage, but the former is rare. A few diarrheic bile-covered discharges give warning of a coming enteric storm. Soon colicky pains and tenesmus set in, which in their intensity are all out of proportion to the small liquid discharges. The attack is sometimes preceded by bilious vomiting and abdominal pains. The stools, which at first were those of simple diarrhea, after three or four days show the presence of mucus and blood. This may continue for four or five days without interfering with the general condition of the patient. When the ulcerations of the colon mucosa begin, the patients are tormented by very severe colicky pains, rumbling, and tenesmus. J. F. Gemmel (*l. c.*, p. 22), in the "Dysentery of the Insane," calls attention to the fact that this class of patients very frequently may not show any evidence of abdominal pain, tormina, etc., which he explains by the benumbing influence of the specific poison and vitiated intestinal secretions on the sensitive nerve-fibers. In amebic dysentery and epidemic ulcerative colitis which I have observed the pain was generally very severe, and probably in Gemmel's cases much of the apathetic condition may be attributed, no doubt, to the mental condition of his patients.

Fever.—There may be fever in amebic dysentery at the beginning, but it is no constant symptom. When present, it may reach about 104° F., but as the disease continues the temperature generally falls to the normal by the fourth or fifth day. Continuance of the fever is suggestive of secondary involvement of other organs or of intestinal lesions of exceptional severity. Rapid fall of the temperature may be regarded as a good indication: the majority of cases that show this sign terminate favorably. Whenever the temperature remains at 102° F. and higher for more than a week or ten days, the prognosis must become guarded.

Evacuations.—The evacuations are small in amount, like gruel or entirely liquid, presenting the yellow color of bile, and containing yellow-stained lumps of flocculent material. There are chiefly two kinds of stools in amebic dysentery, one type of which consists of blood and mucus alone, and another kind of stool which shows matter of two distinct characters—that is, it is either fecal, composed of yellowish-brown material, or, at the same time, a bloody mucous material can be found in it. As the disease progresses the stools assume the appearance of meat-juice, containing yellow, glassy, flocculent material or gelatinous lumps resembling the spawn of frogs. Occasionally the evacuations consist of pure blood, which is dark red, or, if decomposed, presents a chocolate-colored appearance. There is no typical stool for this kind of dysentery. The description of Woodward (*l. c.*) gives evidence that the character of the stools may vary greatly, according to the degree of alteration which the mucosa of the colon has undergone. The number of the evacuations is also variable. In the beginning, or diarrheic stage, there may be five or six evacuations in twenty-four hours. When the stools are dysenteric and bloody from the very beginning, they are more numerous and generally number from ten to twenty in twenty-four hours. Kartulis mentions a case in which there were over thirty passages in one night. The amount of actual fecal matter in these stools is comparatively small—sometimes, though great efforts at expulsion have been made, the patient presses out nothing but a little blood. In his elaborate account J. J. Woodward (*l. c.*, p. 353) considers the constituents of the stools which are of interest in the following order: (1) Feculent matters; (2) mucus, pus, and various admixtures of the two; (3) blood and bloody serum; (4)

portions of pseudomembrane and of necrotic intestinal tissues; (5) bacteria, rhizopods, amebæ, and other parasitic forms; (6) accidental matters derived from the food and medicines taken. Of these ingredients of the evacuations, those in group (2) are the principal ones observed in simple catarrhal dysentery and in the early stages of the diphtheric variety. Those under (4) are characteristic of diphtheric dysentery. Those under (5) are characteristic of amebic dysentery.

As I have already stated, *fecal matter* is absent from the stools, as a rule, after dysentery has become well established. This is particularly so if the disease was preceded by diarrhea, but if it was preceded by constipation, hardened scybala which have been impacted in sacculi of the colon may, during the progress of the disease, be discharged from time to time. When improvement occurs, either temporarily or permanently, fecal matter again makes its appearance in the stool, although it may have disappeared for a time, so that one may regard the reappearance of feculent matters as an indication that the violence of the disease has abated at least for a time. It is, in my experience, very rare to observe normal functioning of the stomach and upper part of the small intestine during dysentery. I have examined the gastric contents of four cases of amebic dysentery and after a Ewald test-meal, and found absolute loss or suppression of gastric secretion in all of them eight days after the onset of the disease. The following were the results by Töpher's test: Free HCl absent; combined HCl absent; total acidity on the average 10; pepsin and chymosin absent; pepsinogen and chymosinogen present. I also examined the vomit in six cases of idiopathic ulcerative colitis during the common epidemics of dysentery occurring in Baltimore. This vomit occurred after bread and water had been given to the patients. The results were approximately the same as stated above in amebic dysentery. It should be mentioned, however, that these analyses were made in each case when the disease was at its climax, and the results agree well with those of Riegel found in acute febrile infectious diseases and also by Hans Herz ("Störungen des Verdauungsapparates," u.s.w., S. 118). In my opinion the suppression of gastric secretion in these cases is due to an acute sympathetic gastritis, because the vomit always contained much mucus, and a few fragments of gastric mucosa found in the vomit showed intense small round-cell infiltration. Oppler ("Deutsch. med. Wochenschr.," 1896, No. 32) and Boas ("Darmkrankheiten," *l. c.*, p. 137) have found this same condition to exist in chronic diarrhea. The presence of undigested food particles, when it occurs in dysenteric stools, is due to two altered conditions in the digestive process: In the first place, the peristalsis is immensely accelerated, thus not giving time for any digestive process to take place; secondly, the digestive secretions of the stomach are most probably suppressed and those of the pancreas also seriously interfered with. To this condition—the appearance of undigested food in the stools—Hippocrates has applied the term lientery (λειεντερία, from λεῖος, smooth, slippery, and ἔντερον, intestine).

A. Schmidt ("Deutsch. med. Wochenschr.," December 7, 1899) noticed in the feces of certain patients small remnants of what looked like wheat, which, on examination, were found to consist mainly of fat and connective tissue, and rarely were there any traces of muscle-fiber contained in the particles. In artificial digestion tests it was discovered that the connective tissue of meat was digested much more rapidly than the muscle tissue in artificial gastric juice, but in a solution of trypsin this relation was reversed: the connective tissue was but little affected and the muscle was rapidly digested. On the ground of these investigations Schmidt concluded that masses of undigested connective tissue in the feces indicated a disturbance of stomach digestion. After giving a test-meal of 100 gm. of scraped or ground roast meat to patients, if he found that an excess of connective tissue was in the stools, he took it for an indication that the gastric functions were disturbed. The condition of the gastric digestion, as shown by samples of this same test-meal drawn from the stomach, is not clearly stated, but the presence of undigested muscle-fibers in the stool is an indication of disturbance of intestinal proteolysis or defective pancreatic digestion (see Chap. IX, Examination of Feces).

As the gastric juice does not digest nuclear tissue, the presence of nuclei in the connective-tissue remnants is regarded as an indication of disturbance in intestinal digestion. Applying the deductions of Schmidt to a large number of stools of dysentery, I have observed, in a number of cases, that they contain meat in which both the connective tissue and the nuclei, as well as the muscle-fibers, are undigested. It is, therefore, reasonable to conclude that both the pancreatic and gastric digestion is seriously interfered with. That the hepatic secretion is also deficient at the height of dysenteric attacks is evidenced by the absence of bile and bile pigments from the stool and the clay or ashy gray or whitish color of the stools. In some cases of dysentery in which bloody mucous stools alternate with the acholic stools the liver may have suspended its functioning as a consequence of direct intoxication of the hepatic tissue, or as a result of overexertion of the organ in its struggle with micro-organisms. As a result of this damage to the hepatic tissue its metabolic and secretory function may become seriously interfered with. Hans Herz (*l. c.*, p. 138) even admits the possibility of paralysis of the liver as a result of dysenteric infection. We shall speak of grave interference with the function of the liver which occurs in dysentery later on. The reaction of the purely dysenteric stools is generally alkaline to litmus and phenolphthalein.

Pus and Mucus.—Of these the mucus is naturally the first to appear. It is present at the beginning of the attacks, and is of a glairy, translucent, sometimes yellowish, appearance, resembling frog spawn or egg-albumen. At the onset of some dysenteries the mucous masses are so coherent that any attempt to remove particles for microscopic examination with a pair of forceps draws the mass out into strings, and a pair of scissors may be necessary to obtain the desired particle. As the disease progresses the mucus becomes opaque, grayish, or yellowish. It may lose its stringy quality, and finally become miscible with water. The various admixtures of mucus and pus in such stools have given rise to the designation mucous, mucopurulent, and purulent stools. The presence of pus in the evacuations is an indication that ulcerations have developed. It is not always possible to recognize pus in the stools with the naked eye. Small quantities of pus can be detected only by the microscope, but purulent stools, whether the pus is visible to the eye or not, have a very characteristic, penetrating, fetid odor. All such stools as we have just referred to contain white blood-corpuscles in varying frequency. Mucus is characterized by its cohesive stringy quality, its reaction with dilute acetic acid (precipitating the mucin), and its solubility in dilute alkalis. The characteristic of pus is its microscopic appearance, presenting numerous corpuscles floating in an albuminous serum.

Blood and Bloody Serum.—Blood may make its appearance in the stools during the course of a variety of diseases. It is observed in diarrhea, hemorrhoids, ulcers of the duodenum, cancer, and tuberculosis of the intestines and in some types of obstructions. It is known to pass the entire bowel after it is swallowed consequent to epistaxis, hemoptysis, or any diseases of the pharynx, esophagus, or stomach that give rise to hemorrhage. When the source of the blood is in the upper part of the digestive tract,—the pylorus, stomach, duodenum, or ileum,—the blood is much altered in appearance by the time it reaches the rectum. This is partly due to the influence of the digestive secretions and partly to that of the bacteria. The blood of this origin is generally of a black-brown appearance, giving the stool a tar-like consistency. Such discharges of blood from the bowel are not frequently observed in dysentery, where the source of the hemorrhage is in the colon, and the blood can usually be recognized by its bright red color. The proportion of blood in dysenteric stools is not so large as it would seem from the quantity of the bloody liquid passed. The bulk of such evacuations is due to a serous transudation issuing from the walls of the colon and lower ileum in this class of diseases. At the beginning of dysentery the serous transudation contains but little albumin, but as the disease advances the percentage of albumin increases. The loss of albumin and albuminates by such stools occurring in dysentery has been quantitatively determined by Osterlen ("Zur Chemie der Ruhr," Henle's "Zeitschr. f. rat. Med.," Bd. VII, S. 253, abstracted by J. J. Woodward, *l. c.*, p. 359). According to this investigator, the average daily loss of albuminates by the stools in the first fourteen days of moderate dysentery is from fifty to sixty grams. The total quantity of material passed by the stool ranges from 1500 to 2000 c.c. daily. In the following eight days after this the daily discharges by stool range from 800 to 1000 c.c., containing on an average twenty grams of albuminates. He estimates the total loss by stool during such a dysentery of three weeks' duration at 30,000 c.c., containing from 900 to 1000 gm. of albumin. Niemeyer ("Lehrbuch d. spec. Path. u. Ther.") has confirmed the opinion held by others that large quantities of albumin may be precipitated by nitric acid in the matters filtered from dysenteric stools, although the dejections are scarcely tinged red and but very few red blood-corpuscles can be found by the microscope. It is concluded, therefore, that the albumin is not derived from the blood mixed with the stools, but is due to a genuine transudation of the blood plasma, which, in Woodward's opinion, may be so considerable as to be largely responsible for the production of dysenteric collapse, which is more strikingly observed in Asiatic cholera. The alkaline reaction of dysenteric stools is also attributed to this transudation of blood plasma, but is, no doubt, in part also due to alkaline fermentations occurring in the intestinal contents. According to Bamberger (Virchow's "Handb. spec. Path. u. Ther.," Bd. VI), dysenteric stools frequently contain carbonate of ammonia.

Pseudomembrane and Necrotic Intestinal Tissue in the Stools.—These characteristic constituents of the dysenteric stool occur when the disease has advanced either to the stage of croupous exudation, which has been described in the section on pathology, or to the stage of ulceration. The appearance of these fragments was supposed to be characteristic of diphtheric dysentery. This is true only of the fragments of pseudomembrane of fibrinous exudation. A necrosed intestinal tissue, however, is found in the discharges of endemic or amebic dysentery, when the autopsy shows no diphtheric deposit. I have already dwelt upon the pseudomembranous deposit and fibrinous exudation infiltrating the mucosa and submucosa, and forming eventually a layer of variable thickness upon the internal surface of the colon. The layers of the normal colon beneath the pseudomembrane to a certain extent undergo necrobiosis, whereby the diphtheric layer becomes separated from the still healthy subjacent tissues. Finally, the pseudomembrane, together with the necrotic intestinal epithelium and submucosa, sequestrates and is discharged in the evacuations. Sometimes the base of the ulcers is in the muscularis mucosæ, sometimes in the mucosa, and sometimes in the submucosa. At places the pseudomembrane alone may separate, leaving the intestinal surface epithelium in comparatively normal condition. Destruction of the normal intestinal tissue does not occur to any great extent in simple catarrhal dysentery, but in all the other forms—the diphtheric colitis, the severe catarrhal colitis, or the ulcerative colitis—more or less destruction of tissue is observed. There are, however, cases of diphtheric dysentery of such extreme intensity that death occurs before the diphtheric layer is sequestrated. The pieces vary in size from that of a small coin—say a one-cent piece and even smaller—to several inches in length. Generally they do not exceed the length of half an inch. The variety of diphtheric stool most commonly observed is composed of bloody serum with numerous fragments of diphtheric and necrotic mucosa floating in it. The macroscopic appearance of these lesions can be seen in the photographs of diphtheric dysentery on the accompanying plates. (Fig. 23 *et seq.*)

These fragments may, on microscopical examination, prove to consist simply of the diphtheric deposit on the surface of the true mucosa, being in that case composed of the diffusely fibrillated or granular fibrin, containing blood- and pus-corpuscles either disseminated throughout or collected into small groups here and there. In case the fragments are genuine diphtheric sloughs involving the intestinal wall, the characteristic anatomical structures of the mucosa and submucosa, the glands of Lieberkühn, etc., are recognizable. The histological elements are considerably altered by the infiltration coincident with the inflammatory process and the necrobiotic changes which occur in the sloughs before they were thrown off. Occasionally it has been established that portions of sequestrated membrane presented the unmistakable histological characters in one part, while in another these elements were entirely obliterated—nothing could be made out but a granular decomposing mass in the place where undoubtedly the true mucosa had once existed. The character and intensity of the retrogressive changes in the sequestrated fragments are no reliable indications of the severity of the disease. The most advanced putrefaction is met with in the so-called gangrenous stools. They are of a greenish or blackish color, composed mostly of a slimy fluid of an extremely putrid odor. The fragments in them are entirely necrotic.

In another chapter I have considered the so-called membranous colitis, sometimes improperly called membranous dysentery, the stools of which may, in some cases, be possible sources of confusion if epidemics of dysentery of the diphtheric variety exist contemporaneously. This disease has sometimes been designated as tubular diarrhea, but has had a large number of designations applied to it (see chapter on Colica Mucosa and Membranous Enteritis). It is most frequently observed in neurasthenic and hysterical patients afflicted with constipation. The attacks which precede the discharge of the mucous masses are accompanied by colicky abdominal pains, distention of the abdomen, and evidences of dyspepsia. The membranous substance is discharged in the form of tubes, shreds, or flat membranes, and generally in considerable quantity. The discharge may even be accompanied by feculent, watery, bloody, or mucopurulent stools. The membranous substances discharged in this form of colitis are composed chiefly of thickened mucus, which dissolves freely in a solution of potassium hydroxid, but does not dissolve in dilute acetic acid. The membranes, when examined microscopically, consist of a hyaline or slightly granular substance, containing a few cells resembling white corpuscles. Occasionally they contain the columnar epithelial cells of the surface epithelium of the intestines. The stools of diphtheric dysentery, as has been emphasized repeatedly, are composed of granular fibrin.

Bacteria, Rhizopods, Amebæ, and Other Parasitic Forms.—These have already been considered at length in discussing the etiology and pathology. Of the bacterial forms, I would repeat here that the bacillus of Ogata, Shiga, Goodliffe, S. Flexner, probably all four identical, and the Bacillus pyocyaneus have been specially associated as causes of dysentery. The Amœba coli is the most important of the protozoan causes of dysentery. Of the other protozoa, the following have been known to cause enteritis and at times dysentery: Trichomonas intestinalis, Circomonas hominis, coccidia, Balantidium coli, Megastoma entericum. Quincke ("Berlin. klin. Wochenschr.," November 20, 1899, No. 47) described a case in which the coccidia apparently produced an enteritis; another in which the symptoms resembled pulmonary and intestinal tuberculosis, but at the autopsy ulcers were found in the colon, and at the bottom of these, numerous amebæ. The balantidium may cause a dysentery similar to amebic dysentery. The Distoma hæmatobium of Bilharz has already been referred to; also the species of Anguilulla intestinalis, which Normand discovered in the stools of

Cochin China dysentery, and which he assigned as the cause of this disease. His results have been disputed by later observers.

Accidental Matters, Ingesta, and Foods in the Stools.—A number of articles of diet can give color to the dysenteric stools; among these are spinach, milk, and coffee. Subnitrate of bismuth and most of the other bismuth compounds give the stool a brownish-black color. Iron preparations color the stool a greenish-black. Copper salts produce a dark-green color. The yellowish-brown or greenish-brown color which results from the administration of calomel was formerly attributed to an increased secretion of bile caused by this drug, but it is really due to the formation of sulphid of mercury by the action of the hydrogen sulphid in the intestinal canal. The literature on the effects of calomel and mercury compounds on the quantity of bile secreted has been reviewed by J. J. Woodward (*l. c.*, pp. 274 to 277). The most recent literature on this subject has been abstracted by Franz Pfaff and A. W. Balch ("Conditions Influencing the Secretion and Composition of Human Bile," "Jour. of Exper. Med.," vol. II, p. 49). The evidence points to the conclusion that calomel, so far from increasing the secretion of bile, actually diminishes it. All the possible ingredients of the diet may be found in the dysenteric stools. This has already been referred to in the paragraph on lientery. Among the substances which have caused especial interest are small semitransparent masses, resembling grains of boiled sago or frog spawn. Bamberger, Griesinger, and Finger, also more recently Heubner, supposed that they originated in the solitary follicles of the colon and were, therefore, indications of the existence of a follicular colitis. Virchow, however, pointed out that if these bodies be treated with iodin, they acquire a blue color, suggesting that what had been called "sago-like mucus" by the above observers, was much more frequently "mucus-like sago." Another constituent of the stools which has caused much discussion as regards its origin is fat. The origin of the fat in the dysenteric stools is, no doubt, that of the fat ingested with the diet. In my own cases at least I have been able to cause the disappearance of fat in the dysenteric stools by excluding fat from the diet.

Concluding herewith the consideration of the stools of dysentery, let us resume once more the general symptomatology.

Nausea and Vomiting.—Frequently the attack is preceded by vomiting; the vomitus may or may not contain bile. Some cases begin with symptoms closely resembling cholera morbus—*i. e.*, vomiting and purging, which, if both persist and the stool assumes dysenteric characters, rapidly end the life of the patient. In another group of cases the vomited matter contains no bile, but consists almost entirely of a colorless or Rhine-wine colored liquid resembling serum. The evacuations are also accompanied by simultaneous purging. At times vomiting occurs only after food or drink has been taken, and is controllable by dietetic methods; but in other cases regulation of diet has no such influence. I look upon the vomiting in dysentery as due very frequently to a sympathetic gastritis coincident with the early stages of the disease. I do not wish to assert that I have seen this vomiting only in the early stages, for it frequently sets in also at a later period of the disease, and in patients who did not vomit at the beginning; nor do I mean to assert that the sympathetic gastritis need be of an identical nature pathologically as the process in the colon. There is also a form of vomiting which gives the impression of an intense and sudden autointoxication. It occurs at the beginning of the disease, and the evidences of accumulation of toxic products are present simultaneously in the urine. Efforts have been made to distinguish clinically between vomiting that is due to gastric irritability and that which is due to actual gastritis. These efforts, in my opinion, if persisted in, constitute a useless waste of time. The practitioner will not go amiss if he treat all such cases as if due to gastritis.

Tormina and Tenesmus.—It is reasonable to suppose that the sensory nervous plexuses in the walls of the colon are more or less involved in the general inflammation of the colon in various types of dysentery. This would make it intelligible that a given degree of intestinal contraction would produce more pain than in cases where the intestines are not inflamed, but as the spasmodic contractions of the muscular coat of the inflamed colon are much more intense than normally, it is natural to expect severer pains to accompany these contractions. We are unconscious of the normal intestinal contractions, but when they are of exceptional violence, we may become painfully aware of them even though the coats of the intestine are normal. An example of this condition is furnished by the griping pain due to advanced intestinal flatulence. The tormina of dysentery are generally due to spasmodic contractions of the muscularis of the colon, and are likely to be followed in that case by an evacuation giving temporary relief, but the tormina may also have their seat in the small intestine, and in that case some time will elapse before the pain is followed by a stool. The Greek physicians (Alexander, of Tralles, Lib. VIII, cap. 9, 1556, edition Bâsel) attributed importance to the time which elapsed between the occurrence of the tormina and the occurrence of the evacuation in diagnosticating the part of the intestine which was ulcerated in dysentery. However, it is very evident, from what we have said concerning the pathology of this disease, that the consideration of tormina and the time of the evacuation thereafter is no positive indication of the locality of intestinal ulceration. The evacuations depend too much upon the character and consistence of the intestinal contents and the normal or pathological location of the colon. By tenesmus is meant an exaggeration of the expulsive efforts of the colon and rectum. Comparatively slight inflammation of the mucosa of this portion of the intestine will promptly give rise to dysenteric symptoms. For a long time tenesmus has been regarded as the pathognomonic symptom distinguishing diarrhea and dysentery. This opinion is held in Woodward (*l. c.*, p. 338), but even the ancients avoided the error of supposing tenesmus to be a symptom of dysentery alone, and enumerated various local causes, such as rectal ulcers, fissures of the anus, hemorrhoids, ascarides, stone in the bladder, and tumors in neighboring organs, by which tenesmus may be produced independently of dysentery. When the mucosa of the sigmoid and rectum is inflamed, the irritability is so much increased that very small quantities of putrefactive feces are sufficient to cause violent tenesmus. The putrefactive material thus expelled readily produces septic processes in the neighborhood of the anus, and the intense expulsive efforts occasionally bring about prolapse of the anus. The perineal abscesses which have been described in association with dysentery are most likely to occur in patients who have previously suffered from hemorrhoids. Such abscesses, when occurring in the sequence of diphtheric dysentery, have been known to take on a gangrenous character and cause extensive sloughing of the perineum before death. By the continued straining, expulsive efforts, and pressure upon the sigmoid flexure and rectum the expulsive muscles gradually become exhausted, so that toward the close of fatal cases tenesmus and tormina, which may have been very severe, may suddenly cease.

Digestion and Utilization of Food in Dysentery.—This has already been considered in the paragraph on the character of the evacuations. Not only are the digestive secretions very much reduced in their normal proteolytic, adipolytic, and amylolytic ferments, so that food transformation can not take place properly, and in severe cases complete annihilation of the power to digest proteids and fats occurs, but, in addition to this, the bacterial, putrefactive, and fermentative processes already coincident with the disease attack the undigested food and thereby add to the already existing disintegration. Uffelmann (" Deutsch. Arch. f. klin. Med.," Bd. XIV, S. 228) has pointed out that in dysentery accompanied by continuous fever and dry mouth and tongue, the saliva becomes very scanty and is acid in reaction, and is no longer capable of transforming starch into dextrose. The points just considered indicate the main object of treatment—viz. (1) a proper method of nutrition and (2) control of the septic processes in the intestine.

Strangury and Tenesmus of the Bladder.—When the disease is fully developed, the urine becomes more and more concentrated, of a high specific gravity, and more and more acid in reaction. At the same time the vesical mucous membrane in the vicinity of the neck of the bladder becomes hyperemic or even actually inflamed. As a result of this, vesical tenesmus of such intensity has been known to occur as to require the use of the catheter for its relief. According to Finger (" Prager Vierteljahresschr. f. d. prakt. Heilk.," 1849, Bd. IV), this vesical inflammation may extend into the ureters and reach the pelvis of the kidneys. When the attack of inflammation of the bladder is mild, it promptly yields to the use of alkalis or remedies which dilute the urine. As these may, however, increase the diarrhea, it is sometimes advisable not to give alkalis, but to treat the vesical complication locally by irrigations, etc. In women catarrhal and diphtheric inflammations of the vagina and uterus have been observed.

The Urine in Dysentery.—The urine is scanty, highly colored, and highly acid. On cooling, it deposits a sediment of urates.

When the disease is accompanied by cystitis, the evidences of this inflammation are found in the urine—viz., mucus, mucopus, and leukocytes. Albumin and indican are frequently present. Woodward (*l. c.*, p. 386) holds that when albumin is present in the urine of a patient suffering from acute dysentery, it will be found that he has previously presented symptoms of chronic disease of the kidneys. This does not conform to my experience. I have personally observed albumin in the urine of three young patients, aged fourteen, eighteen, and nineteen, who, upon analysis of the urine, never gave any evidence of renal disease prior to the dysentery. The relation of intestinal diseases to abnormalities in the urine has been considered in a separate chapter by Dr. H. Adler. In all such relations we must not only consider the kidney and the renal circulation, but, above all, the condition and composition of the blood, for there can be no doubt that the kidney often functions only as an excretory organ for albuminous bodies which are not in adequate chemical form for the blood. So much at least is conceded for the excretion of the albumoses, the presence of which in the urine is looked upon as an abnormal composition of the blood, not as disease of the kidney.

Complications.—Various complications may develop in the course of endemic dysentery. They occur either as the immediate consequence of pathological processes in the colon or they are caused indirectly by the absorption of bacteria or toxins from the intestine and lymph-channels. Among the direct consequences are the gangrenous and necrotic processes of the colon, producing sphacelus of entire sections of this organ in extreme cases; furthermore, typhlitis, perityphlitis, intestinal perforation, and consequent peritonitis. Among those complications caused by the absorption of intestinal bacteria, parasites, or toxins are the liver abscesses, inflammation of the parotid gland, abscesses of the brain, lungs, and spleen, the arthropathies, or pseudorheumatic affections, and various forms of paralysis and neuritis.

Peritonitis as a Complication.—Woodward (*l. c.*, p. 388) describes a form of peritonitis developing from an exaggerated hyperemia of the peritoneum, corresponding to those parts at which the diphtheric process is most intense or at which the ulceration has penetrated most deeply. When intestinal ulceration has progressed beyond the circular muscular fiber of the intestine, and is accompanied by a plastic effusion tending to produce adhesions between the affected portions of the colon and abdominal walls or adjacent viscera, it is sometimes very difficult to ascertain, even microscopically, whether perforation has actually taken place or not. Woodward's remarks would suggest that peritonitis is possible without perforation. He describes this as most prone to occur in the cecum, but it may happen in any part of the colon. During life the process is characterized by exquisite tenderness along one or more points in the course of the colon, and a steady pain, which is felt even in the interval between the paroxysms of tormina. Perforation may occur in any portion of the colon, but at autopsies is most frequently demonstrated in the cecum. It is often accompanied by abscess formation, perityphlitis, which may discharge into the intestine and in rare cases externally through the abdominal walls. In chronic dysentery peritonitis may give rise to considerable serous effusion, which occasionally remains in the form of ascites, even after convalescence has occurred. Clinically peritonitis as a consequence of perforation can not be differentiated from the general peritonitis not accompanied by this accident.

Intussusception and Ileus.—At autopsies on subjects dead from dysentery intussusception and ileus (intestinal obstruction) are occasionally observed, and as they are frequently unaccompanied by the evidences of inflammation, they have been attributed to spastic conditions occurring in the death agony. But occasionally it occurs in dysenteric cases during life, giving rise to the characteristic symptoms of intestinal obstruction. Woodward does not report any such cases as having occurred in the dysenteries of the Civil War. As the fecal discharges are suppressed in dysentery and the dysenteric discharges themselves consist only of mucus, pus, blood, and bloody serum, intussusception or other form of obstruction, if it occurred, would not interfere with the discharge of these materials from portions of the intestine located below the point of obstruction. The other symptoms of obstruction—viz., obstinate constipation, vomiting, abdominal tenderness, pain which is both continuous and spasmodic—are not sufficiently characteristic for a diagnosis, as they occur also in dysentery. The symptom of constipation loses its diagnostic value, for the reasons just stated.

Intestinal obstruction occurring as a complication of dysentery would, therefore, probably escape recognition during life. There are rare cases, however, in which the intestine caught in the intussusception sloughs away and is discharged by the evacuations during the life of the patient. The occurrence of the symptoms above mentioned during an attack of diarrhea would be much more likely to attract attention than when they occur during dysentery, for in that case the symptom of constipation would be more characteristic (see literature on the subject by J. J. Woodward, pp. 390 and 391).

Abscess of the Liver (*Hepatitis Suppuratita*).—This is one of the most frequent complications of endemic, amebic, or tropical dysentery. In epidemic dysentery hepatic abscesses are rare. Kartulis (*l. c.*) has collected 4790 cases of dysentery reported by various clinicians in tropical countries, among which were observed 957 liver abscesses. According to these statistics, this complication occurs in 20 per cent. of the cases of tropical dysentery. The same author has tabulated 474 cases of epidemic dysentery, in which occurred thirty-six hepatic abscesses—about 7.6 per cent.

The following statistics, compiled by the author, are of interest in this connection. They are for the largest part included in the statistics of Kartulis:

CASES OF ABSCESS OF THE LIVER ASSOCIATED WITH DYSENTERY IN INDIA.

AUTHORS.	AUTOPSIES ON DYSENTERY.	LIVER ABSCESS.
Ballingall, "Practical Observations on Fever, Dysentery, and Liver Complaint in India," Edinburgh, 1818, ...	35	4
Annesley, "Diseases of India," London, 1828, ...	51	26
Waring, "An Inquiry Into the Statistics and Pathology of Liver Abscess," Trebandrum, 1854, ...	259	69
Eyre, "Medical Notes on Dysentery," "Indian Annals of Med. Sci.," October, 1855.	118	27
Ranking, "Statistical Gleanings from the Records of the Second Madras European Light Infantry for the Years 1840 to 1860 Inclusive," "Madras Quar. Jour. of the Med. Sci.," vol. VII, 1863, ...	140	41
Moore, "The Relation of Dysentery to Abscess of the Liver," "Trans. of the Med. and Phys. Soc. of Bombay," vol. VIII, 1862, ...	494	90
Macpherson, "On Bengal Dysentery," Calcutta, 1850, ...	293	46
Chuckerbutty, "Cases Illustrative of the Pathology of Dysentery," "Indian Annals of Med. Sci.," vol. XIX, 1865, ...	39	3
	1429	306

CASES OF ABSCESS OF THE LIVER ASSOCIATED WITH DYSENTERY IN ALGIERS.

AUTHORS.	AUTOPSIES ON DYSENTERY.	LIVER ABSCESS.
Catteloup, "Recherches sur la dysenterie du Nord de l'Afrique," "Receuil de Mém. de med., etc., Militaure," VIII, 1851, . .	240	47
Mouret, "De la coincidence de l'hepatite ou des abscess du foie avec la dysenterie dans le pays chauds," Paris, Thesis No. 52, 1853, ...	761	133
	1001	180

In rare cases the dysenteric hepatic abscesses are associated with pulmonary and cerebral abscesses, but these complications occur only in the endemic form. Councilman and Lafleur observed six cases of hepatic abscess among nine cases of amebic dysentery. W. T. Howard, Jr., and C. F. Hoover ("Amer. Jour. of the Med. Sci.," August and September, 1897) observed three cases of hepatic abscess in four cases of dysentery. Two of the hepatic abscesses communicated with the right pleural cavity through the diaphragm. These authors give an explicit account of the etiology, symptomatology, and pathology of hepatic abscess. According to Rouis, 15 per cent., and according to Chvostek, 25 per cent., of these hepatic abscesses are painless. The amebic or tropical abscess is located, in the majority of cases, in the posterior or superior portion of the liver. Vomiting, icterus, and ascites are comparatively rare complications. Involvement of the peritoneal covering of the liver gives rise to a friction which may be palpable as well as audible. The friction sound is heard in the axillary line, between the eighth and tenth ribs, and must be differentiated from the sound which may be caused by pleuritic inflammations. The diaphragm may be involved, and through inflammation it may lose its muscular contractility, become attenuated, and no longer resist the negative thoracic pressure and the aspiration of the lungs. When the lungs retract, the diaphragm and the liver follow upward. This is followed by the clinical sign of dyspnea and cyanosis. In rare cases it may become important to distinguish between infraphrenic and supraphrenic location of pus. For this purpose it has been suggested that a trocar be inserted into the region made out as being affected with the abscess. If the pus flows out during inspiration, it is infraphrenic; if during expiration, it is

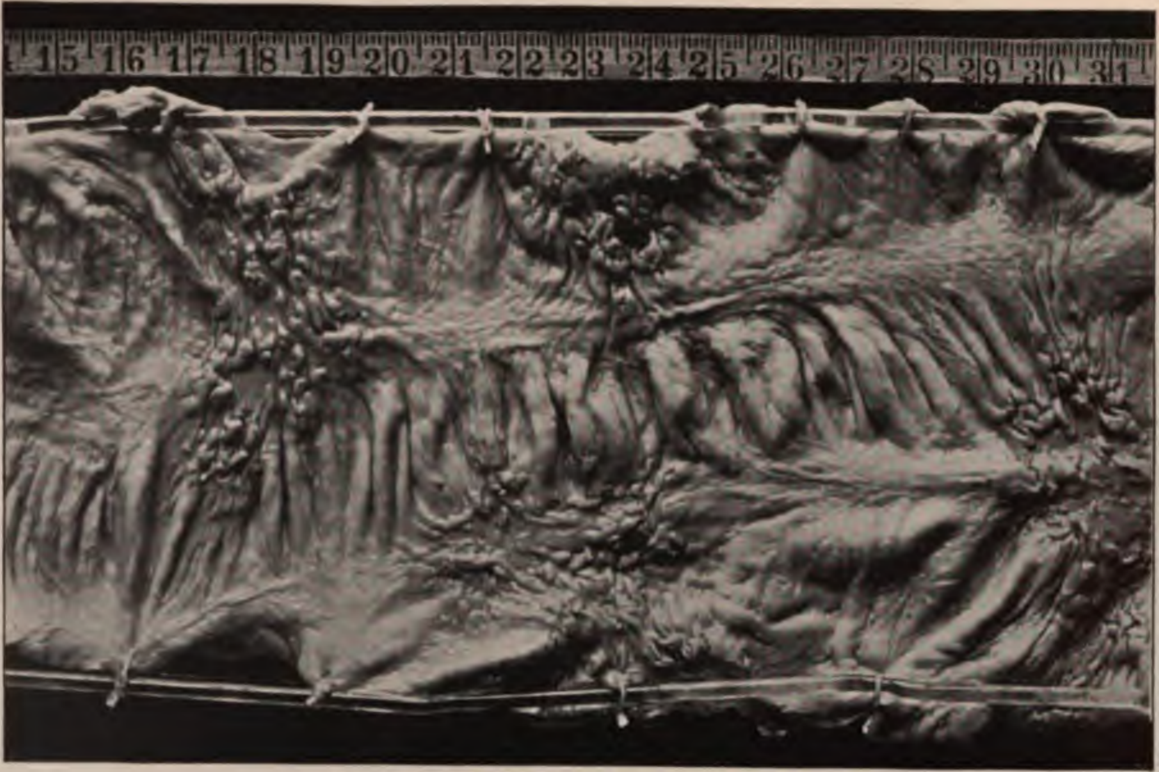


Fig. 25.—Healing Dysenteric Ulceration of Colon.

supraphrenic or intrathoracic. This test is not always reliable, for adhesions between the liver and the diaphragm, or between the pleura and the diaphragm, may frustrate the clinical value of this sign, as was shown in case 2 of Howard and Hoover (*l. c.*), where the pus flowed out during expiration and the abscess was, nevertheless, in the liver, or infraphrenic.

Councilman and Lafleur found the Amœba coli in the pulmonary abscesses, and Kartulis found them in the abscess of the pleural spaces after perforation of the diaphragm from a liver abscess. Abscess of the spleen has been observed only once by Kartulis. In 80 cases of idiopathic ulcerative colitis J. F. Gemmel (*l. c.*) has observed but 2 liver abscesses. In 156 cases of acute or chronic inflammation of the intestinal mucous membrane without ulceration (Woodward, *l. c.*, p. 548) there was but 1 case of hepatic abscess, but in 115 cases of diphtheric dysentery there were 3 cases of single abscess and 4 of multiple abscesses. Adding these and a few other stray cases together, Woodward collects 14 cases of multiple abscess and 9 of large single abscess in 511 cases of flux in which the intestine was ulcerated, and but a single case of large hepatic abscess in 156 cases of flux in which the intestine was not ulcerated. Woodward gives also an excellent compilation of statistics and clinical reports on hepatic abscess up to the time of his publication.

Inflammation of the Spinal Cord and Nerves as Complications.—The arthropathies and pseudo-rheumatism are complications of dysentery which are occasionally met with after the violence of the attack has been spent. But I have seen two cases of epidemic dysentery which were preceded by pseudo-rheumatism in all the joints five days before the dysenteric attack manifested itself. If the cases are slight, the pains are generally felt only in the muscles of the abdomen, back, breast, and thigh. Sometimes the pains are localized in one joint. The large joints, the knees especially, are most frequently affected. Pseudo-rheumatic affection of all the joints—the so-called polyarthritis pseudo-rheumatica—is, fortunately, a rare occurrence, and observed only after extremely severe attacks of dysentery. We have no means of telling whether or not a given case of dysentery will be followed by arthritis. When once developed, however, it has, in my experience, proved a long-continuing and wearisome complication. Cardiac complications have so far been rarely reported in this connection. The prognosis of this complication is, as a rule, favorable, as most cases recover without any ankylosis.

Myelitis and Neuritis.—Damaschino, von Hoffmann, and Laveran have reported complications of myelitis following dysentery, and Delioux de Savignac ("Traite de la Dysenterie," 1863) has observed two cases. As a rule, this complication expresses itself in the form of paraplegias or hemiplegias of the extremities, which are either complete or partial, and may be accompanied by pains or be painless. A curious observation has been made in connection with these nervous lesions, and that is that the dysenteric symptoms may suddenly disappear with the onset of the paralysis. The occurrence of paralysis in connection with dysentery as an occasional complication was known to Galen, who attributed it to the injudicious suppression of the discharges by rash medication. S. Weir Mitchell ("Paralysis from Peripheral Irritation," etc., "New York Medical Journal," February, 1866) states that he has seen *many cases* of paraplegia following upon dysentery, acute or chronic. In the statistics given by Woodward on this subject (*l. c.*, p. 410) we find only one case of paralysis following acute dysentery, and nine other cases following in what were reported as chronic diarrhea. The statistics of this part of Woodward's magnificent report seem unreliable, owing to the defective clinical reports from which he had to make them up. One can not fail to gain this impression in view of the above remark of S. Weir Mitchell concerning the greater frequency of this complication. The observations of this scholar were made in the Army Hospital for Diseases of the Nervous System in Philadelphia.

Some of the cases of this complication which have been reported in literature are probably due to lead-poisoning—probably from the use of acetate of lead in the treatment of the disease. If the patients are thoroughly exhausted from the dysentery, the prognosis of the myelitis is unfavorable, but if they have sufficient vitality, recovery is probable. Nothing positive is known of the pathogenesis of this complication: it may be due to direct infection, to autointoxication, to trophic influences, or it may be, as Leyden holds, a genuine neuritis. Personally I have observed two cases of neuritis following exceptionally severe dysentery. In one male patient the neuritis seemed to involve only the sensory fibers of the right leg, causing intense neuralgia, which lasted fourteen days, than ceased in that limb and appeared in the left leg.

Symptomatology of Other Endemic Forms of Dysentery.—Among the other forms of endemic dysentery, reference must be had to the dysentery of Cochin China, of Japan, and of the Philippine Islands, and other forms which have been described by English physicians in India under the name of *white flux,* white purging, or *hill diarrhea.* According to Fayir, it is identical with the chronic diarrheic or dysenteric form of Cochin China. Under the name of "sprue," a form of dysentery occurs in Java and China which, according to Moursou, is identical with the white flux. Rasch ("Virchow's Archiv," 142) has described it as a chronic diarrhea associated with chronic gastritis, atrophy of the intestinal mucosa, and contraction of the liver. It is not definitely settled whether this affection is a chronic dysentery or an independent disease. There can be no doubt that chronic diarrhea and dysentery are two entirely different diseases. Complications of the two may occur, and then they may not be so easily distinguishable clinically. Kartulis thinks that sprue is an independent disease, not a dysentery. Finally, we will briefly refer to a disease of the colon which is known as the diarrhea of pilgrims, and which was first described in reports of quarantine physicians from ports along the Red Sea. Occasionally it presents the form of severe diarrhea; at other times, of chronic dysentery. Severe cases have even been confounded with cholera. The patients come mainly from the pilgrims which have returned from Mecca. In 1893 there were 1940 of such pilgrims, according to Zachariadis, received into the hospital of Tor. Of these, 1601 were suffering from gastro-enteritis, and many of these had genuine dysentery. Kartulis and Kaufman consider that the pilgrim's diarrhea is a similar disease to the Cochin China diarrhea or to the white flux, but that among those afflicted there are many who have the typical amebic dysentery.

To revert once more to the symptomatology of epidemic dysentery, we may briefly say that in the beginning it is very much like that of the endemic or amebic dysentery. As the disease progresses, however, it may show essentially different symptoms from the endemic form. The light cases of epidemic dysentery have been spoken of as catarrhal, and the grave cases as diphtheric or croupous.

Catarrhal Stage of Epidemic Form.—The disturbances of the general condition of the body are more pronounced and more regularly present than in the endemic form. This stage may exhibit a variety of clinical pictures, at times so characteristic that they have been described as separate types of the disease under the name of *rheumatic*, *bilious*, and *choleriform dysentery*. The *rheumatic* variety is a dysentery associated with symptoms of rheumatism, localized in one joint or attacking several; the muscular and articular pains are probably due to autointoxication. The *bilious* form is characterized by bilious vomiting, fever, headache, and icteroid pigmentation of the skin and conjunctivæ. The *choleriform* type occurs most frequently in diphtheric and croupous dysentery. The prodromal stage presents irregularities of the stools, anorexia, nausea, and vomiting. Diarrhea and general weakness start the disease, which is then rapidly associated with colicky pains, thin but still fecal stools, and violent tenesmus. In the beginning the number of the stools is from four to six in twenty-four hours, but they increase in number and offensiveness according to the intensity of the disease. The tongue may be dry or very much coated. The pulse is small, compressible, and averages about 100 beats in the minute. Frequently there is strangury. On palpation and percussion nothing can be ascertained but sensitiveness to pressure in the left iliac fossa and in the umbilical region. The transverse colon may be distended and tympanitic. After a duration of from one to two weeks the cases recover, generally without complications. The stool, which is at first like gruel and gives evidence of the presence of bile constituents, but of a very fetid odor, later on becomes watery, almost odorless, and containing a few clumps or flocculent material. Microscopically the stools show, in addition to undigested food remnants, bacteria, blood elements, epithelial and pus-cells, intestinal epithelium, but no amebæ.

Diphtheric or Croupous Stage of Epidemic Form.—This stage of epidemic dysentery is characterized by frequent evacuations accompanied by tenesmus and great abdominal pain. The temperature is normal or subnormal, and the pulse may become so soft and

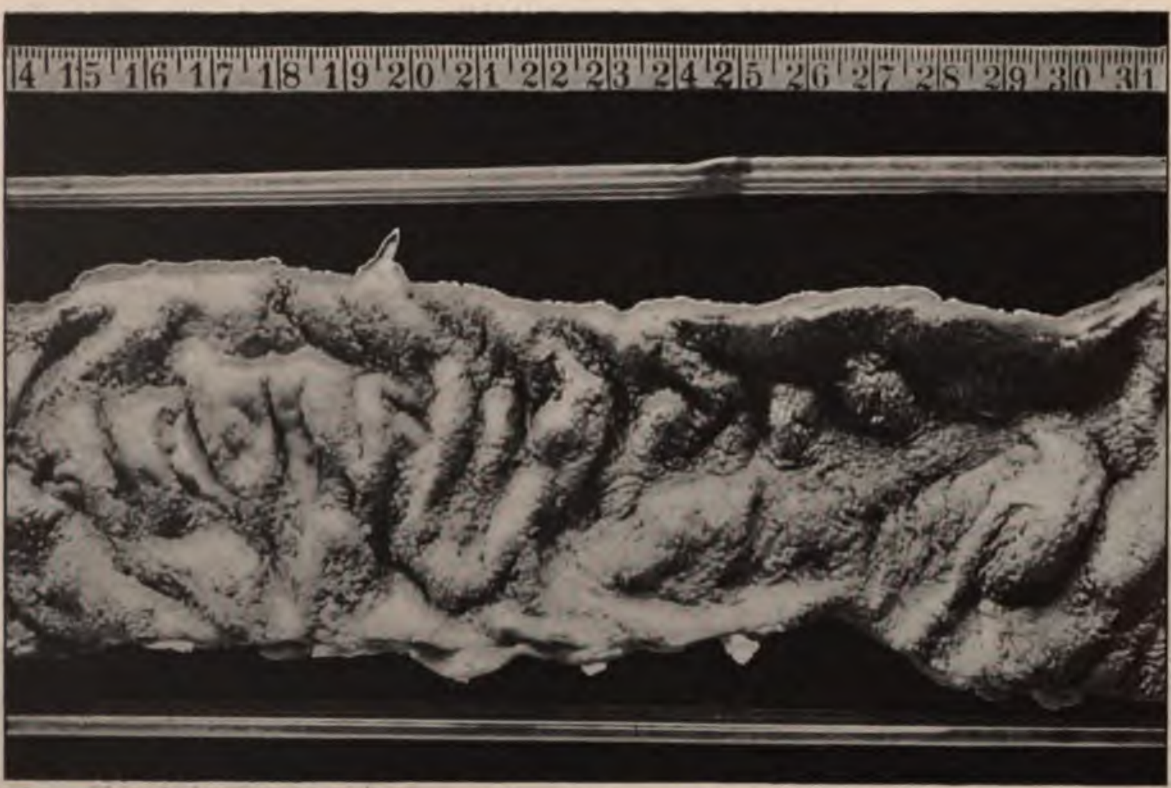


FIG. 26.—DIPHTHERIC DYSENTERY (COLON).

compressible that it frequently can not be felt at all. Palpation, auscultation, and percussion elicit nothing but great distention of the abdomen, which is very sensitive to pressure. The other abdominal organs outside of the intestines do not give evidence of any special involvement. The stools are of a bloody red color in the beginning, but later on are dark, and even black, generally containing patches of necrotic mucosa. They are of an extremely gangrenous odor. Microscopically they contain enormous masses of microbes, cellular detritus, pus, blood, and necrotic tissue.

The various forms of epidemic dysentery doubtless have a different etiology, and it is natural, therefore, that they should present a variety of clinical pictures. In some epidemics this picture is that of simple catarrhal dysentery of a benign type and short duration. In others, however, we are confronted with the grave symptoms of diphtheric, croupous, or gangrenous colitis. These latter forms may eventuate into the so-called typhoid or choleriform dysentery. In the *typhoid* forms we may have the clinical picture of enteric fever, with the very intense symptoms—exceptionally high temperature, delirium, unconsciousness, and coma. Even bed-sores and cutaneous hemorrhages have been described in this variety of epidemic dysentery. The choleriform variety runs its course under the clinical picture of cholera.

Prognosis.

The prognosis of uncomplicated dysentery is, as already stated, favorable. Flint has proved that this type may terminate in recovery spontaneously, but in the diphtheric or croupous stages the prognosis is grave. In cases in which gangrene has supervened the outlook is hopeless. Death may occur from sepsis, exhaustion, hemorrhages, intestinal perforation, peritonitis, etc.

CHRONIC DYSENTERY.

Both endemic as well as epidemic dysentery may become chronic, but this is especially so of the endemic form, or amebic dysentery, which, under certain conditions, may result in an exceptionally long continuance of the ulcerative process, with little or no tendency toward healing. Chronic dysentery is particularly frequent after military encampments and in large institutions, which is probably attributable to the fact that the causes which led up to the original attack are generally persistent under such conditions, and constitute a never-ceasing danger of infection. We shall refer to this again in discussing Prophylaxis. The causes of chronic dysentery are, in various cases, improper diet or return to the diet which caused the original attack of the disease—infected drinking-water, neglect, incorrect therapeutic management, particularly the abuse of mercury, and in rare cases, perhaps, an individual predisposition. Chronic dysentery presents, in the majority of cases, the symptoms of a severe chronic enteritis, and with alternating improvements and aggravations it may extend for months and years. The slightest dietetic errors or exposure to cold seemingly bring on a new attack. The patients emaciate to skin and bones and are psychically very much depressed, but may be able to follow their usual occupations with few interruptions. Other cases of chronic dysentery are continuously compelled to remain in bed. Such cases emaciate very rapidly and appear like living skeletons, with immensely distended and tympanitic abdomen. The transverse colon is often palpable, by reason of its increase in thickness, due to the chronic inflammatory process. Death may occur from asthenia, cardiac failure, marasmus, perforation, peritonitis, or abscess of the liver. Recovery, however, has been observed even in severe cases. The evacuations in chronic cases occur without tenesmus, and with but very slight pain. They are generally liquid, of a yellowish-green or blackish-brown color, containing blood, mucus, and pus. The liver is the most frequent organ that is involved secondarily in the process. This seems to have been a rare complication in the autopsies that came under the observation of J. J. Woodward (*l. c.*), for he gives only 4 cases of abscesses of the liver in 156 cases of acute and chronic catarrhal inflammation of the intestinal mucosa, and 115 cases of diphtheric dysentery; whereas the British writers whom he quotes (*l. c.*, p. 549) give 90 hepatic abscesses in 494 autopsies. The liver is generally hypertrophic and painful on pressure, even if there be no hepatic abscess. There is generally a bronchitis, and the heart-sounds are difficult to make out by auscultation. It is in chronic dysentery that the condition known as lientery, which I have elsewhere described, is most frequently seen, and characterized by the undigested condition of the food as

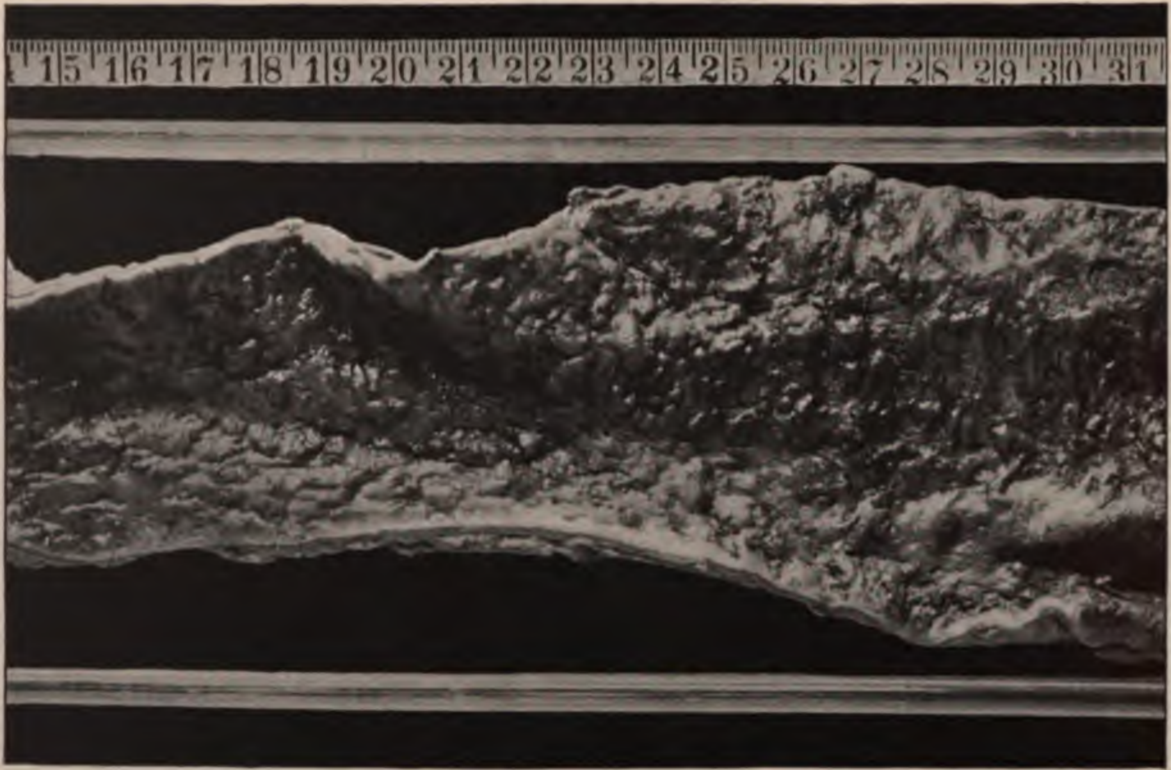


FIG. 27.—CHRONIC DYSENTERIC ULCERATION OF COLON.

recognized in the stools. There are mainly three varieties of chronic dysentery:

Variety 1.—The symptoms of a typical attack of dysentery after an attack of average intensity do not cease entirely, but become milder, the whole disease assuming a gentler type. This continuance of symptoms occurs in such as may be the subjects of individual predisposition, and who during the entire time of chronic dysentery maintain well-regulated habits of life. The chronicity extends from two to four months, but is eventually arrested.

Variety 2.—An acute attack of dysentery ceases entirely, but a relapse occurs during convalescence, which, after varying periods of time, may show alternating improvements and aggravations. Long-continued attacks of diarrhea may alternate with periods of obstinate constipation. Emaciation and hepatic complications are frequent if the disease is neglected.

Variety 3.—This variety begins under the guise of a surreptitious catarrhal diarrhea, the dysenteric nature of the disease remaining hidden or unrecognized until the inflammatory process in the colon has assumed an ulcerative type. In this variety evacuations of an ordinary diarrheic character alternate with typical dysenteric stools —*i. e.*, fetid, bloody, and mucous masses, accompanied by tenesmus. After from two to three months of this process, general marasmus and exhaustion follow.

Pathology of Chronic Dysentery.—Woodward includes under chronic dysentery all fluxes observed during the Civil War among the troops with the exception of those resulting from tubercular ulceration of the intestines. Anatomically the chronic fluxes might be naturally divided into two great groups: (1) Those resulting from chronic inflammation of the mucous membrane of the intestines with or without ulceration; (2) those resulting from large unhealed ulcers left after the separation of diphtheric sloughs. The symptoms of both these types of chronic dysentery, as well as the treatment to be pursued, are so similar that the doubtful attempt to establish clinical distinctions based upon these anatomical grounds would serve no useful purpose. Similarly, a subdivision of these cases into chronic diarrhea and chronic dysentery, in accordance with the presence or absence of tenesmus, pus, mucus, mucopus, or blood in the stools, would not be found to correspond with anatomical classification. In chronic fluxes following acute dysentery the tenesmus very often ceases after the separation of diphtheric sequestrations, so that this is not a distinguishing symptom. Concerning the occurrence of intestinal strictures and stenoses as a result of contraction of dysenteric ulcers, to which attention was first called by Rokitanski ("Oester. Jahrb.," Bd. XVIII, 1839, S. 13). Woodward states that of the entire enormous clinical and pathological material collected in his work, not one case has been reported to the Surgeon General's office either during the war or since, and the Army Medical Museum does not possess a single specimen of stenoses from this cause. This is an important denial of the frequency of intestinal strictures resulting from dysenteric ulcers, an opinion held by a number of German writers (see "Strictures of Intestines," Chap. XXV).

The intestinal lesions of chronic dysentery may be arranged in three groups, which may exist either singly or variously combined: (1) Chronic inflammation of the mucosa or submucosa without ulceration; (2) chronic inflammation associated with follicular and other ulcers of the colon, at times complicated with a pseudomembranous exudative inflammation between the ulcers; (3) extensive ulcerations of the large intestine as a consequence of sloughing during a previous attack of acute diphtheric dysentery. In the chronic inflammation without intestinal ulceration curious cyst-like formations are met with in the submucosa, varying in size from $\frac{1}{10}$ to ¼ of an inch in diameter. They contain a gelatinous, thick, or mucoid fluid. Woodward (*l. c.*, p. 512) has given an excellent chromolithograph of these gelatinous cysts of the colon in chronic dysentery.

Follicular and Other Ulcers.—In the preceding I have already given a description of the histological character of the ulcers of amebic dysentery. Concerning the ulcers of chronic dysentery, I should add that they are always accompanied by thickening of the mucosa and submucosa, and sometimes of the remaining layers of the colon. This hypertrophy of the layers may assume such dimensions as to cause a contraction of the colon,—a narrowing of its lumen,—giving it a hard and rigid feeling. This condition may extend over the entire colon or be limited to certain parts. The submucosa sometimes, on sections cut perpendicularly to the mu-

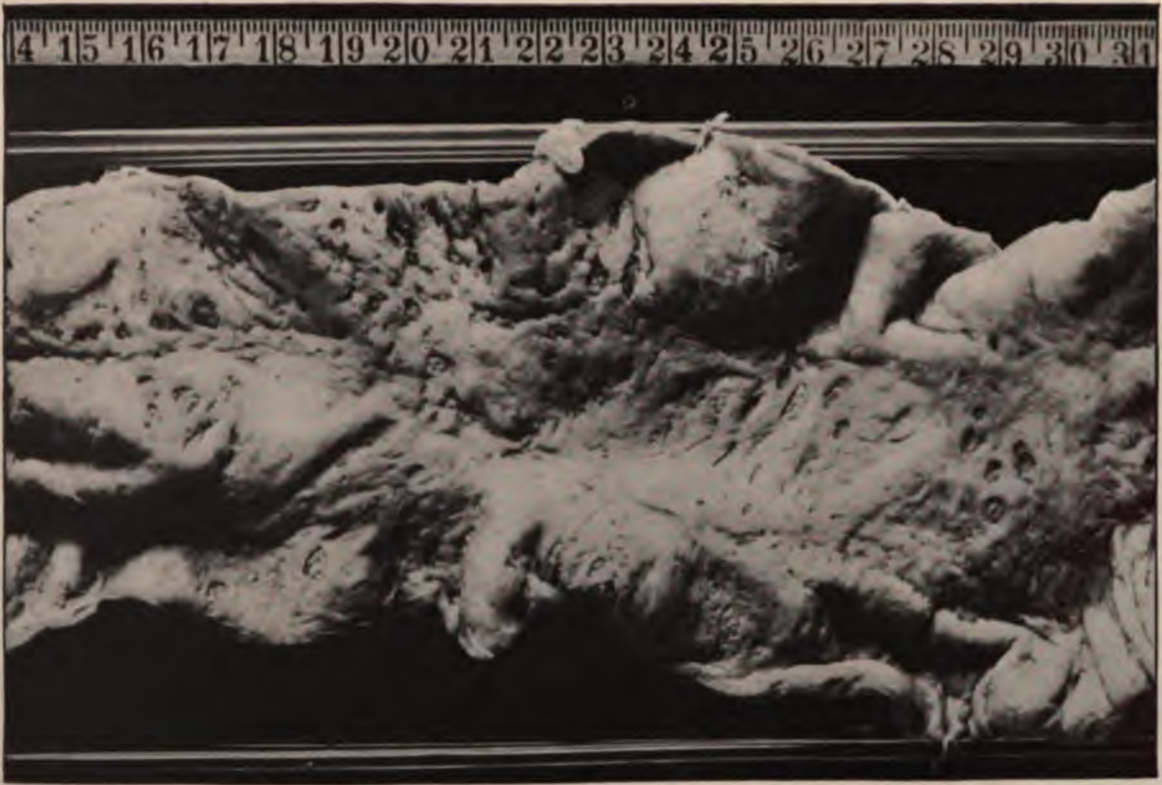


Fig. 28.—Dysenteric Ulceration of Colon.

cosa, presents a very characteristic whitish, semitranslucent appearance. The ulcers may originate in the follicles, or independently of them in the mucosa between the follicles. The follicular ulcer appears first as a superficial erosion destroying the true mucosa only, but later on extending into the submucosa. This kind of ulcer is at the beginning quite small, so that at the autopsy numerous small isolated depressions or losses of substance are noticeable where the follicular ulcers existed. One of the plates in this chapter illustrates such follicular dysentery; it was taken from a specimen in the Pathological Museum of the Johns Hopkins Hospital, through the kindness of Professor Wm. H. Welch. When several follicular ulcers coalesce, they form a loss of substance very similar to the separation of a diphtheric slough. The ulcers caused by coalescing of several follicles present overhanging edges, from the fact that they spread more rapidly in the submucosa than in the mucosa. Councilman and Lafleur, after an examination of the specimens of Woodward in the Army Medical Museum, arrived at the conclusion that many of these cases were typical amebic dysentery. Both forms of ulcers, the follicular and the nonfollicular variety, may permeate all the layers of the colon, and eventually even cause perforation. In a pathological specimen the follicular ulcers appear as small, rounded, or ovoid depressions, frequently giving the impression as if they were punched out. The nonfollicular ulcers appear as irregular erosions of the mucosa of varying size, and, like the follicular ulcers, they lead to destruction of the true mucosa and submucosa. The edges of many of the ulcers of both varieties are sometimes fringed with the pseudomembranous deposit. The diphtheric process frequently supervenes and complicates the follicular ulceration; thus every phase of the diphtheric inflammation can be observed along with the characteristic follicular ulcers.

The degree to which we can recognize the nature of the primary lesion depends upon the stage of the process at the time of death. The diphtheric inflammation sometimes proves fatal so promptly that only a slight deposit between the ulcers is recognizable; in that case we should be justified in concluding that the ulcers were primary. In other cases, however, the patients survive until the fully formed pseudomembrane has sloughed; in that case it is impossible to ascertain which is the primary lesion. Under favorable circumstances the ulcers of both varieties may heal, giving rise to peculiar stellate cicatrices in the case of the follicular ulcers. In fatal cases such healed ulcers or cicatrices are found at intervals between unhealed open ulcers.

Treatment.

The treatment of dysentery may be considered under three headings: (1) The prophylaxis; (2) the dietetic treatment; (3) the physical and medicinal treatment.

The Prophylaxis.—Among the most well-recognized causes of dysentery are: Infected drinking-water; infected, improper, unwholesome, or even excessive amount of food; excessive use of vegetables and fruit or such as have come in contact with infected water; abuse of alcoholic beverages; improper or insufficient clothing; neglect of disinfection of fecal evacuations. Neglect of such factors would sufficiently explain the occasional prevalence of dysentery in military encampments, etc., besieged cities, and large institutions. Whenever a large number of human beings are compelled to live together under similar circumstances, the water should be sterilized either by boiling or satisfactory filtration. Fruits and vegetables that are necessary for food should never be washed in the water in ordinary use, but only by boiled water. Strict supervision of the cooking department and the cooks themselves by the physicians in charge is indispensable. The cooks of large encampments or institutions should be compelled to keep themselves in as aseptic a condition as possible. The physician himself should be chief of the kitchen. The hands and forearms should always be disinfected before preparing the articles for food, and no individual at all diseased should be permitted to assist in the kitchen. In summer the windows of the kitchen should be screened to prevent the entrance of flies and other insects. All indigestible food must be avoided, but particularly so in the tropics. Even moderate use of alcohol is unhealthy in tropical regions. Much of the dysentery during times of war is attributable to improper selection of the army rations and to improper preparation of the articles constituting these rations. Those interested in the rations of the American and foreign armies can instruct themselves concerning them by reference to W. Gilman Thompson's work on "Practical Dietetics," page 680. It may be

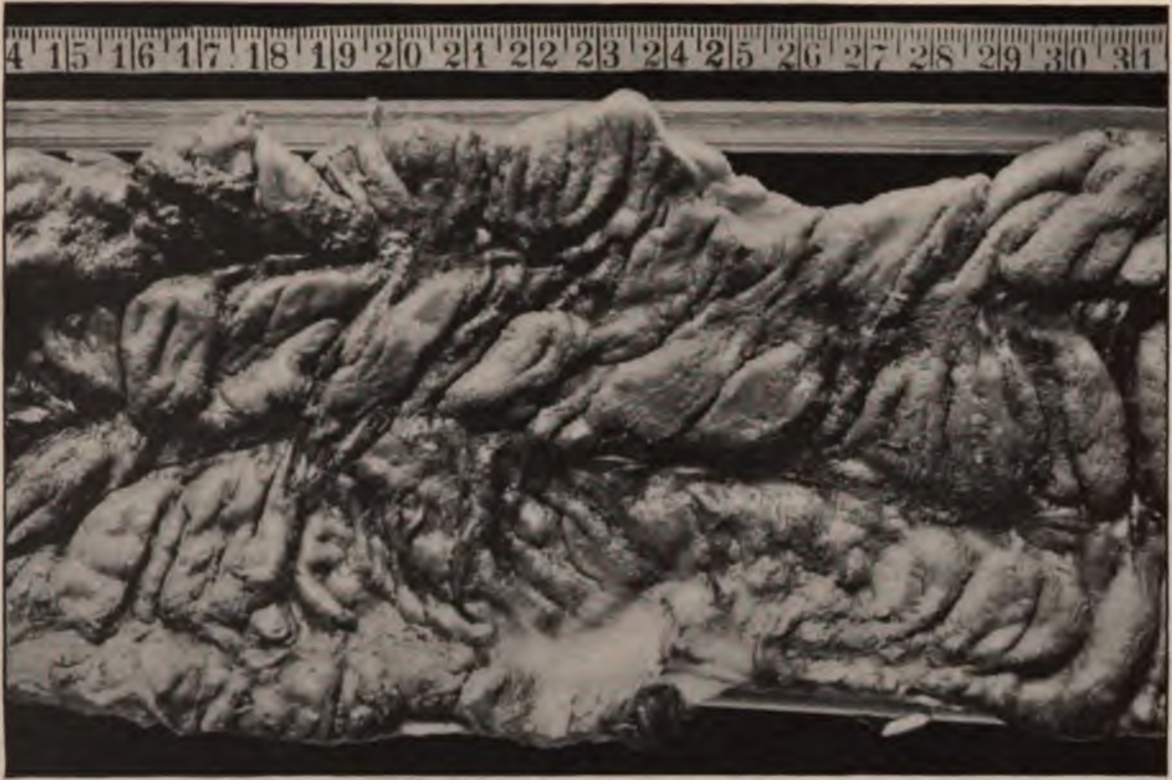


FIG. 29.—DIPHTHERIC AND HEMORRHAGIC COLITIS.

pointed out here that the principles laid down by Captain Chas. E. Woodruff, Assistant Surgeon U. S. A. ("Journal of the United States Cavalry Association," March, 1894), are questionable, particularly the reference that economy is essential. In view of the fact that dysentery kills more soldiers than bullets, the policy of economy in providing the cheapest kind of food for the soldiers should be emphatically condemned—it belongs to the "penny wise, pound foolish" school of economy, which sees no further than its nose, tries to save in the expense of the food of the soldier, and swells the pension roll by assigning contracts to the lowest bidder in many instances without providing for the most rigid sanitary and chemical control and inspection of the food-supplies. On the other hand, some of Captain Woodruff's principles seem sufficiently logical to recommend their general execution—for instance, that the food of the soldier must approximate the food of the nation at large, so that on enlistment there will be no rapid change of diet that will impair health.

Diet and Nutrition in the Tropics.—It has been supposed that much less food is required in the tropics than in the temperate zone, because metabolism and tissue waste are much less active in warm temperatures: Thus Maurel ("Du règime alimentair physiologique," "Le Bulletin medicale," 1899) recommends 1.5 gm. of nitrogenous or proteid food to a kilogram of body-weight in temperate climates. The ratio of the nitrogenous food to the non-nitrogenous should be as 1 is to 4; the amount of fat should be one gram to a kilogram of body-weight. The remainder should be given in carbohydrates. Maurel believes that neglect to reduce the amount of food in hot seasons of the year and also in the tropics is responsible for a variety of digestive disturbances, because the excess of nutritive material is liable to decompose, and harmless intestinal parasites may, by this decomposition, be converted into pathogenic forms. Hypernutrition, according to this, forms a favorable culture-medium in the intestinal canal for the development of pathogenic bacteria. Accordingly he recommends for the tropics a diet in which the nitrogenous constituents amount only to one gram to a kilogram of body-weight, with the carbohydrates and fats in the same proportion as above stated. In individuals who have to perform heavy work in the tropics he concedes an increase in the nitrogenous portion of the food of one-fifth of the above amount, and of the nonnitrogenous portion an increase of two-fifths of the above amount.

The investigations of C. Eijkmann ("Virchow's Archiv," Bd. CXXXIII, S. 111) demonstrate, however, that the working-classes in Batavia consume an amount of food equal to 2470 calories for the Europeans, and 2512 calories for the Malays, so that there is no reduction in the amount of food practicable in the tropics if work is required of the individual. It is worth considering also that it is not the temperature of the air in the tropics which controls metabolism, but the temperature of the body as maintained by the clothing. The artificial temperature produced by our clothing is veritably a tropical one in all zones, compelling us to exist at a temperature of 34° C. on the average.

Concerning the statement of Eijkmann (*l. c.*) that the relations of nutrition and the amount of food of European and Malayan working-men in the tropics do not differ essentially from those of Europe, which is followed by the statement above given that 2470 calories is the total amount of food energy they take in, it may be stated that the standard given by Molleschott for a man at moderate work is 3160 calories; that given by Wolff, 3030 calories; that by Playfair, 3630 calories. Atwater's caloric standard for a man at hard work exceeds 4000 calories ("The Chemistry and Economy of Food," United States Department of Agriculture, Bulletin No. 21, p. 208). In our country the standard for food requirements for laborers is higher than in Europe, the plane of living of the European working-man being low in comparison to that of the people of the United States. The figures of the authors last quoted, compared to those of Eijkmann, permit of the conclusion that, after all, the diet in the tropics has been unconsciously, perhaps, reduced, because experience has taught that less food is required than under similar conditions in the temperate climate.

This experience I have personally made on myself while traveling in the tropics, and have felt better and was actually capable of greater physical endurance when the diet was reduced to about 2500 calories.

The Clothing.—A frequent error made by inhabitants of the temperate zones when traveling in the tropics, and also in the

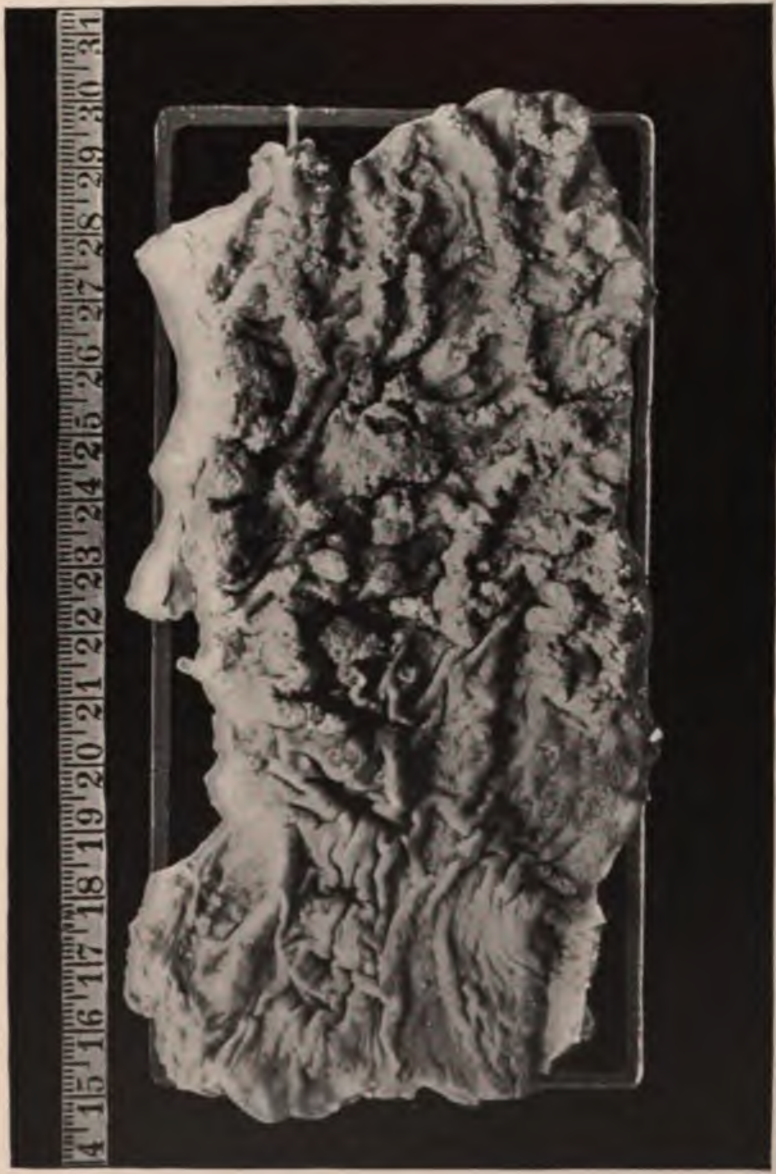


FIG. 30.—DIPHTHERIC COLITIS.

equipment of troops intended for military operations in the tropics, is that the clothing does not afford sufficient protection against the frequent and brusque differences of temperature and against the influence of atmospheric moisture in tropical countries. The nights in the Philippine Islands, as well as in South Africa, are frequently very cold—at least from 15° to 20° C. colder than the day temperature. The vicissitudes from meteorological changes are particularly great during the rainy season.

Alcoholic Beverages.—Alcoholic beverages act unfavorably upon the digestive and nervous systems in the tropics. When there is need for stimulation, they are expediently replaced by coffee, chocolate, tea, or small and regular doses of strychnin.

Disinfection of Fecal Evacuations in Encampments, etc.—This is an unsolved problem, especially when large numbers of human beings are forced to live together, as in large institutions and army encampments. The gravity of the subject justifies, in the first place, the closest and severest supervision of the sinks, latrines, etc., by the military and medical authorities. The present method of disposing of fecal evacuations in encampments does not appeal to my judgment as being the best that can be enforced. It must be especially insufficient in tropical climates. I refer to the digging of a shallow pit or hole into which the fecal matter is discharged, and at intervals covered with chlorid of lime or slaked lime. This can not even approximately disinfect the mass of fecal matter that accumulates, which must inevitably pollute the atmosphere by its gaseous products, and the soil and drinking-water by the draining. I would suggest the plan of maintaining constantly a large fire at some distance from the camp of each regiment, where each soldier on having an evacuation should be compelled to take it up upon an iron shovel, and personally destroy it in the flames. In certain places where wood is abundant, as near forests, this would be not nearly so expensive as the use of chlorid of lime, but even where wood is difficult to obtain, the greater expense of the plan would be far outweighed by the greater security of the hygienic environment to the camp.

Dietetic Treatment.—Few diseases are so readily amenable to treatment in their incipient stages as acute dysentery. On the other hand, there is no more difficult disease to manage dietetically or medicinally than chronic dysentery. A frequent cause for regret is the experience that dysenteric patients rarely seek medical aid immediately after the first symptoms of the disease, a circumstance which is particularly dangerous in endemic or amebic dysentery, because this form readily becomes chronic and leads to complications. According to Griesinger, the mortality of this form of dysentery in Egypt amounts to 36 per cent. These facts are also emphasized in the statistics giving the mortality of Europeans living in tropical countries and the mortality among natives suffering from dysentery. Thus, Hirsch and Fayrer give the mortality in India at from 10 to 20 per cent. for the Europeans, but 37.5 per cent. (Roux) for the Hindus. The mortality among the English soldiers suffering from dysentery in India is 1.5 to 5.84 per cent. The mortality of the epidemic form in Europe is from 3 to 10 per cent.*

Special Diet.—The objects to be accomplished in the diet for dysenteric patients are threefold: (1) To support the strength and nutrition of the patient; (2) to offer no food that will increase the intestinal putrefaction; this can be accomplished by the exclusion of indigestible food, which in this case embraces all solid diet; (3) food that will at the same time appease the intense thirst of the patient. The most useful dietetic article that answers to all these indications is milk, which should always be given after it has been sterilized or Pasteurized. In my experience there are very few patients who can not tolerate milk or who have a repugnance to it, and in these cases this can be overcome by the addition of sodium bicarbonate or lime-water. C. A. Ewald (von Leyden's "Handbuch der Ernährungs-Therapie," Bd. II, S. 264) states that it has been his experience that this article of diet is, as a rule, not available in dysenteries. He argues that it increases the pains and tenesmus and causes fullness in the epigastrium and vomiting, and he holds that milk is proper only during the period of convalescence, and should not be used in the acute period, except as an addition to glutinous soup. I am aware that milk can not be given in certain cases of dysentery, but from a large number of cases treated personally in Bay View Asylum and in private practice in this city and during an epidemic treated at Beaufort, N. C., I consider that these cases are the exception. The milk should be given in a lukewarm condition, and in the beginning of the attack not more than one quart (one liter) in twenty-four hours. This I gradually increase, when it is shown that the food agrees well, to two liters. Sometimes very small quantities of milk—for instance, two tablespoonfuls every fifteen minutes—can be tolerated when it can not be taken otherwise; but I should be loath to torture a patient by giving a tablespoonful of milk every ten minutes until two liters have been taken in twenty-four hours, as has been recommended by Bérenger-Féraud. The addition of coffee, tea, or a little table salt sometimes makes the milk palatable when it could not be taken otherwise. The next most suitable article of diet in my hands has been egg-albumen or gelatin albumin. The white of eggs is beaten up thoroughly, the froth removed, and brandy added in the proportion of one tablespoonful to the whites of three eggs. This can be sweetened to taste with sugar. Gelatin albumin is made by dissolving egg-albumen in liquid gelatin, adding good wine (sherry) or brandy, sugar, and allowing the mixture to congeal. I have discontinued the use of beef bouillon, because in my opinion it contains little nutritious matter, and simply constitutes a suitable culture-medium for the intestinal bacteria. Among the other articles of diet I can recommend the following, the preparation of which is described in the author's work on "Diseases of the Stomach": Chicken jelly (p. 255); egg and Hochheimer (p. 431); arrow-root blanc mange (p. 257); port-wine jelly (p. 257); a number of the gelatinous or glutinous soups to which aleuronat flour (*l. c.*, p. 261) can be added are useful dietetic substances for this disease—for instance, the meat purée soup (p. 262) and tapioca soup (p. 262).

---

* Exhaustive statistics concerning the mortality from dysentery in the various parts of the United States before and during the Civil War can be found in J. J. Woodward's work, *l. c.*

Eggs: Eggs are best given in a soft-boiled condition or raw. They must be allowed only after a cautious test of a half to a whole egg has been made, because they occasionally increase the abdominal distention.

Solid food: It is usually said that the patient may return to the use of solid food when fever, tenesmus, and pain have ceased and the tongue becomes clean, and when the desire for food is expressed by the sufferer. These indications are frequently fallacious, for they do not indicate a complete restoration of the mucosa of the colon to a normal condition. We should, therefore, not return to the use of solid food until the mucopurulent or sanguinolent stools are replaced by normal ones, and then very cautiously, by giving meat purée or raw scraped meat, finely minced breast meat of chicken or pigeons, and tender fish. At this time eggs may also be given with less fear than during the acute stage.

Dysenteric patients generally suffer from intense thirst, but it is imperative to avoid carefully the extremes of temperature in the beverages permitted for the quenching of this thirst. While cold water is much enjoyed by the patient, and may serve to relieve the heat and dryness of the esophagus and mouth, it frequently causes increased diarrhea, pain, and tenesmus. Warm water or water at the room-temperature does not produce these ill effects and is much more rapidly absorbed. Kartulis allows Vichy and other carbonated mineral spring waters. As these can be enjoyed only when they are cold, I should be loath to prescribe them. If the patient can be watched, a glass of cracked ice may be placed where he can reach it, and small pieces allowed to dissolve in his mouth from time to time.

It may become necessary, in the course of the treatment, when collapse threatens from cardiac asthenia or from depression of the nervous centers, to prescribe alcohol in some form. If possible, the imported German and French clarets, rich in tannic acid, should be used in such cases. Assmannshauser and Beaune Burgundy answer best in my experience. The French Burgundies are also very useful at times. A good way is to give the wine mixed with water; in some cases hot wine mixed with hot water and sugar will act more promptly. It is unwise to give alcohol throughout the disease as a matter of routine. At times coffee, tea, and strong bouillon may be used to alternate with the alcoholic beverages in producing the desired stimulation.

Repeated attacks of vomiting after a slight improvement has been manifest are due, in my experience, to the retention and accumulation of fecal masses, to overdrugging, or to dietetic errors. In such cases all remedies should be left aside so far as is consistent with the successful management of the case, and the diet limited to small amounts of rice- or barley-water. Somatose is not applicable to the diet of dysentery, because it increases the diarrhea; but nutrose, eucasin, and sanatogen are permissible, and even urgently indicated in dysenteries that last longer than two weeks and seriously threaten the strength of the patient; they should then be mixed with the glutinous soup, coffee, or gelatin.

Physical Methods of Treatment.—*Lavage of the Colon.*—In view of the localization of the disease in the colon, the direct or topical application of medicated solutions to the large intestine is the most rational plan of treatment, for in this way only can antiparasitic and astringent medications be brought into immediate contact with the diseased mucosa. The method of application is the following: If the patient is strong enough, the knee-chest position is most favorable to the entrance of the solution; but if this position is impossible on account of the prostration of the sufferer, the position first on the left and then on the right side is, in my experience, the next most favorable one, because it facilitates the entrance of the solution into the transverse colon after it has passed the sigmoid flexure. A pillow under the hips will secure the effect of gravitation. The amount that is allowable at first should not exceed one liter, and the temperature, 100° F. The elevation of the reservoir containing the solution should not exceed five feet above the rectum. In acute cases the extreme irritability of the rectum and sigmoid flexure unfortunately causes tenesmus, and sometimes even entirely frustrates any attempt to irrigate the colon. In these cases it is well to anesthetize the rectum with a small quantity of a 4 per cent. solution of cocain or a suppository containing cocain and morphin, and, after a sufficient time has elapsed, the medicated solution can, as a rule, be allowed to flow in slowly. A number of antiseptics, astringents, and anodyne substances have been used in this way. Among these are alum, acetate of lead, sulphate of zinc, sulphate of copper, and nitrate of silver, all of which I have discarded and no longer use.

They are very painful when used in sufficient strength to act as antiseptics, and if used in weaker solutions, they are ineffective. It is unjustifiable to use poisons in this way; thus carbolic acid, bichlorid of mercury, and salicylic acid should be discarded. After a large experience with this form of treatment in both epidemic and sporadic dysentery, I have limited the use of drugs for irrigating purposes to four substances—quinin, tannin, suspensions of bismuth salts, and solutions of ichthyol. The strength of the quinin irrigations is one part of quinin to 2500 of water. They are most effective in amebic dysentery, in which they act similarly to the tannin irrigation, which shall be described presently. Quinin and tannin have the advantage of having been thoroughly tested, and while they are decidedly destructive to the parasites in the colon, they are not poisonous. It is conceivable that the quinin irrigations may become absorbed and produce the characteristic symptoms of cinchonism. But no poisonous effect whatever has been observed by me after many hundreds of irrigations with tannin. A 0.5 per cent. solution of tannin is the proper strength to use. Of this, at first one liter is allowed to enter the colon and permitted to remain there for from ten to fifteen minutes. These irrigations are given three times daily. In amebic dysentery the parasites, as a rule, disappear after the fourth day of irrigation in this manner. It is necessary to continue the treatment for a week after the last evidences of amebæ in the discharges have been detected. In acute dysentery, as a rule, one week of this treatment suffices; then the evacuations become fewer in number, mucus, pus, and blood disappear gradually, and on the fifth day and thereafter there are no passages except after the irrigation.

Enteroclysis, or High Irrigation of the Colon.—This is given similarly to the irrigations just described, except that the method requires a rubber colon tube two meters in length. This tube is introduced into the rectum for about eight or ten centimeters, and if the patient can tolerate it without pain, still further. The patient is placed in the knee-elbow position or on the right side, with knees drawn up, and directed to breathe deeply and regularly during the irrigation. If pain should occur on introduction of the tube into the rectum, or if an obstruction is felt, the instrument should be slowly withdrawn and an attempt made to introduce it without pain by changing the position of the sufferer. It is well to begin the inflow as soon as four centimeters of the tube have entered the rectum, because the outflowing liquid will push away obstructing masses of fecal matter or folds of mucosa. A 0.5 per cent. solution of tannin is as strong as should be used, and the solution should be allowed to remain in the intestine about ten minutes. These irrigations are practised about three times daily, and after improvement has set in, about once daily until the stool becomes normal. A curious observation is frequently made in the effect of these irrigations on the evacuation of the membranes and necrotic tissue in chronic dysentery. Occasionally after the detachment and evacuation of pseudomembranous tissue have ceased entirely, they reappear again during a continuance of this treatment. In my experience it is wise to keep up the enteroclysis until no membrane or tissue of any kind has been seen in the returning liquid for one week. It is sometimes necessary to give additional medicated injections into the rectum or medicated suppositories. Thus, inflammation or ulceration of the rectum (dysenteric proctitis) calls for rectal irrigation with iced water, tincture of opium, or cocain. An injection, with a small syringe, of a suspension of bismuth subgallate and bismuth subnitrate of equal parts has, in my experience, proved decidedly soothing and reduced the tenesmus. The following suppository is a practical one for the symptoms just indicated:

℞.	Cocain sulphate,	3	grains
	Denarcotized extract of opium,	3	"
	Extract of belladonna,	1½	"
	Cacao-butter, enough to make 6 suppositories.		

DOSE.—One suppository every three or four hours.

In any form of dysentery in which there is evidence of persistence of ulcerations of the colon and slow healing in spite of the irrigations just described, I can recommend a trial with irrigations by means of suspensions of bismuth subnitrate and bismuth subgallate, to which ichthyol has been added. The following formula is the one I use most frequently:

℞.	Bismuth subnitrate,	6	drams
	Bismuth subgallate,	6	"
	Ichthyol,	2	"
	Warm water,	1	liter.

DIRECTIONS.—Allow this injection to remain in the colon for fifteen minutes.

Infusions of sterile normal salt solutions may become necessary to combat two distressing conditions: (1) The intense thirst and drying out of the tissues, due to diarrhea and hemorrhages, and (2) the depressing autointoxication. In cases of threatening collapse from heart failure, these infusions can be made into a vein; this will rarely be necessary; as a rule, the infusion into the subcutaneous areolar tissue will suffice. The method, when carried out with strictest aseptic precautions, is very helpful, and in my practice has saved life in two cases of extreme autointoxication from dysentery.

Medicinal Treatment by the Mouth.—There are no drugs which have a curative influence upon the dysenteric process. Kartulis ventures to say that ipecacuanha has an almost specific influence upon the dysenteric process, but he admits that in severe cases and in amebic dysentery it is ineffective. Courtois-Suffit ("Traité de Médecine," de Bouchard et Braissaud, tome IV, p. 559) makes the following statement: "The medicine *par excellence* of dysentery is ipecac; it plays in this disease the same rôle that quinin plays in intermittent fever." Of course, if ipecac plays the same rôle in dysentery as quinin does in the treatment of intermittent fever, the practitioner need have no further worry about the cure of the former disease, because quinin is a positive cure for intermittent fever. I shall return to my personal experience with the ipecac treatment of dysentery later on, but will first describe the manner in which ipecac is usually employed. A preliminary dose of opium is given, or ¼ of a grain of morphin is injected hypodermically, and a turpentine stupe or mustard plaster placed over the epigastrium. Half an hour later twenty grains of powdered ipecac root are given, and this dose repeated once or twice at intervals of an hour. All liquids are forbidden, and only a little ice allowed. In this manner about sixty grains of ipecac can be given without causing emesis. Ipecac is also given according to the Brazilian method, which is principally in vogue among the French colonial physicians. From two to eight grams of powdered ipecac are boiled with 500 gm. of water and allowed to stand twelve hours. This constitutes the first infusion, which is decanted. The residual powder is infused twice more with the same amount of boiling water, each 500 c.c. being permitted to remain in contact with the powder for twelve hours before it is decanted. There are thus three infusions of 500 c.c. each to be taken. The first fluid decanted is administered at once or divided into several portions, and, according to Kartulis, always produces emesis and diarrhea. After taking the second 500 c.c. emesis is not so frequent and the stool occurs at rare intervals. The third 500 c.c. causes some emesis, but no diarrhea. Three days are required for this treatment, the first, second, and third decanted infusions being taken in the order they are prepared. Courtois-Suffit adds that if this treatment does not modify the evacuations, the series should be begun over again.

My own experience with the ipecacuanha treatment of dysentery as observed in the practice of southern colleagues and in ten cases of acute epidemic dysentery at Bay View Hospital has led me to discard it entirely. Very little, if any, appreciable improvement was observable, and in eight of the cases a marked general depression and weakness of the heart supervened after the administration of the drug. In recent literature bearing on this subject, as well as from the observations made with ipecac in the treatment of dysentery during the Civil War (J. J. Woodward, *l. c.*, p. 703), one is justified in concluding that the ipecacuanha method is by no means an efficacious treatment for this disease. I have observed the Brazilian method of giving this drug as it was carried out by a physician in one of our southern States. It made the impression upon me of a barbarous maltreatment. Osler ("Principles and Practice of Medicine," p. 138) states that he has never seen the marked beneficial treatment as described by the Anglo-Indian surgeons. Ipecacuanha and its alkaloids, according to Schmiedeberg ("Grundriss der Arzneimittellehre," S. 115), act as poisons upon the capillary walls, and can also cause inflammation. On the basis of our present experience, and from a critical study of reliable reports thus far obtained, it can not be asserted that ipecacuanha acts beneficially upon the dysenteric process.

Calomel, Castor Oil, and Saline Purgatives.—These medications are practical only when it is possible to treat a case at the onset, or when an individual is in the prodromal stage of the disease, which is occasionally observed in an epidemic. When the bowel has been thus thoroughly cleansed from above, it should be kept as nearly aseptic as is possible under the circumstances by giving nothing but sterilized food and early irrigations of the colon. If calomel is intended to exert a purgative effect, it must be given in large doses,—from six to eight grains at once,—but if an antiseptic effect is desired, it is best to give ½ grain every hour. Kartulis prescribes it in combination with naphthalene in the following manner:

℞.	Calomel,	8 grains (0.5 gm.)
	Naphthalene,	15 " (1 gm.)
	Oil of bergamot,	3 drops.

Mix and make 10 powders. One powder every hour.

Intestinal Antiseptics.—Among the most frequently used are salol, betanaphthol, naphthalin, bismuth subnitrate, and bismuth salicylate. When these drugs are administered by the mouth, their antiseptic action is, in my opinion, very doubtful. These drugs can all, however, be used in the form of an enteroclysis, in which case they act in a surgical manner, being brought directly in contact with the ulcers in the colon. This method of treatment is by far the most efficacious.

Astringents.—These may be of organic or inorganic origin. The organic astringents owe their power largely to the presence of tannic acid, and I have spoken of them in connection with the treatment of enteritis. There are two which seem, when given by the mouth, to have some value in the treatment of dysentery; these are tannigen and bismuth subgallate, each given in ten-grain doses every three hours. Large doses of bismuth subnitrate given by the mouth are useful in conjunction with the remedies above mentioned. Monneret recommended giving as much as seventy grams of this substance a day.

Anodynes and Antispasmodics.—About the only remedies of value in this class are opium and its alkaloid, morphin. These drugs should never be given in large doses, for should they bring on constipation, it is very likely to lead to the formation of metastases and hepatic abscesses. When pain and tenesmus are severe, these drugs should never be withheld, though very frequently intense tenesmus and pain will be relieved by enteroclysis. In several cases of epidemic dysentery in which the pain was very severe and the patient could not take opium because of extreme susceptibility, orthoform injected into the colon by high rectal enema acted very favorably. Orthoform is a drug which acts similarly to cocain, and it must come in direct contact with the ulcers of the colon in order to do good. About one dram of the powder in a pint of water injected high up into the rectum and retained twenty minutes will arrest the pain, provided the colon has been previously evacuated by a cleansing enema. Orthoform has been used in the same manner for the relief of pain from gastric ulcer.

After-treatment.—There are two sequelæ that usually require attention after an attack of dysentery. One is constipation and accompanying intestinal flatulence, and the other is anemia or general debility. For the treatment of the constipation see the special chapter devoted to this subject, but it should be stated that the oil enemata are most suitable in these cases. The anemia and general debility require iron, strychnin, and sometimes quinin. The following formula is suggested:

℞.	Strychnin sulphate,	1/3	grain	(0.02 gm.)
	Ferratin,	1	dram	(4 gm.)
	Essence of calisaya (P. D. & Co.),	1 1/2	ounces	(45 gm.)
	Elixir of gentian,	enough to make 6	ounces	(180 gm.)

M.—One tablespoonful three times a day.

Concerning the use of quinin in medicated irrigations of the colon I have already spoken. This drug is very useful in all forms of dysentery, not only in those occurring in malarial regions. If the temperature be high, five or six grains can be given every three or four hours, reduced to two grains as soon as the temperature subsides. In giving the enteroclysis the patient should be carefully protected from catching cold, and hot-water bottles packed around the gluteal region and on the abdomen as soon as the procedure is over. Similarly, when a patient uses the commode, or night-stool, he should be provided with a pair of woolen stockings and wrapped in a blanket to prevent a chill. If, during the enteroclysis, the high rectal tube does not pass easily, it is by no means rational to use force in its introduction, because of danger of perforating one of the ulcers that may have formed. It is safest to introduce the tube not so far in that case and permit the liquid to seek its own way. Cases without fever and showing an asthenic condition should be kept in bed and packed with warm bottles. The evacuations should be passed into the bed-pan. Cases that have high temperature require occasional sponging off with cold water.

CHAPTER XXI.

ULCERS OF THE INTESTINE.

The intestinal tract presents a larger number and a greater variety of ulcers or ulcerative processes than any other internal organ. It is desirable, for didactic clearness, to separate the ulcers which appear as independent diseases—the so-called primary or idiopathic ulcers—from those which occur as partial or concomitant phenomena of other diseases—the so-called secondary ulcers. Ulcers which appear as independent diseases are: (1) The so-called peptic duodenal ulcer, sometimes called the simple ulcer of the duodenum; (2) ulcerations following extensive cutaneous burns; (3) ulcers due to embolism; (4) ulcers due to toxic causes—for instance, the uremic and mercurial ulcerations.

Secondary ulcerations which occur in association with other diseases of the intestine are: (1) The ulcerative processes which occur in acute infectious diseasesf—or instance, in typhoid or enteric fever, in anthrax, and in dysentery; (2) the ulcerations occurring in chronic infectious diseases—viz., tuberculous and syphilitic ulcers; (3) there are very rare ulcerations of the intestine which occur as a result of infection with favus—in actinomycosis and in infections with Mucor corymbifer; (4) finally, ulcers of the intestine have been observed in certain constitutional diseases, such as acute leukemia, gout, and scorbutus. The entire intestinal tract from the pylorus to the anus is liable to be the seat of ulcer formations varying in their etiology, histology, and general configuration. By general consensus the following ulcerations are generally excluded from a clinical treatise on intestinal ulcers because they are usually considered in special chapters—viz., those ulcerations which have developed as a result of the breaking-down of neoplasms or in consequence of perforation into the intestine of abscesses located outside of it, the entrance of corrosive substances from the stomach and, furthermore, ulcers in consequence of invaginations and incarcerations of the intestine, and those produced by the action of irritants or corrosive substances upon the mucosa of the rectum.

Courtois-Suffit considers intestinal ulcerations according to the following scheme:

A. Inflammatory ulcerations,	Acute and chronic enteritis.		
B. Specific ulcerations,	Typhoid fever, dysentery, anthrax, tuberculosis, syphilis.		
C. Ulcerations due to interference in the circulation,	Embolism	Ulcerative endocarditis. Purulent infection.	
	and Thrombosis.	Arterial Venous.	Thrombosis of the mesenteric vessels.
D. Ulcerations developed from neoplasms,	Cancer of the intestine, leukemia, pseudoleukemia, lymphadenoma.		
E. Ulcerations occurring in amyloid degeneration of the intestines.			
F. Toxic ulcerations,	Arsenic, corrosive sublimate, antimony and potassium tartrate, alcoholism (Bouchard and Brissaud, "Traité de Medécine," second edition, p. 483).		

It is impossible to classify intestinal ulcers satisfactorily both from the pathogenic and clinical aspects. I will consider only such intestinal ulcerations as present a certain independence and individuality in their clinical history. From a clinical standpoint, Nothnagel divides intestinal ulcers into six groups:

I. Ulcerations in consequence of necrobiotic processes.
    1. Simple duodenal ulcer.
    2. Ulcers in consequence of cutaneous burns.
    3. Embolic and thrombotic ulcers; among these are classed the intestinal ulcers peculiar to diffuse neuritis.
    4. Amyloid ulcerations.
II. Ulcerations due to inflammatory processes.
    1. Catarrhal ulcers.
    2. Follicular ulcers.
    3. Stercoral or decubital ulcers.
III. Ulcerations in association with acute infectious diseases.
    1. Typhoid or enteric fever.
    2. Dysentery.
    3. Diphtheria.
    4. Anthrax.
    5. Sepsis.
    6. Erysipelas.
    7. Varioloid.
IV. Ulcerations occurring in chronic infectious diseases.
    1. Tuberculosis.
    2. Syphilis.

V. Ulcerations occurring in constitutional diseases.
    1. Gout.
    2. Scorbutus.
    3. Leukemia.
VI. Toxic ulcerations.
    1. Uremic ulcers.
    2. Mercurial ulcers.

HISTORY.

The first mention of an intestinal ulceration occurs in a book by Jacobus Fontanus which appeared in 1612. The son of this author died from dysentery. The autopsy was made by Bontamper, who demonstrated the existence of more than 200 intestinal ulcerations accompanied by abscess. Morgagni had recognized intestinal ulcers. In 1715 Conrad de Brunn described an intestinal ulceration which, in the opinion of Courtois-Suffit (*l. c.*), was manifestly tuberculous. An excellent monograph by J. Krauss ("Das perforirende Geschwür im Duodenum," Berlin, 1865) contains the literature up to the year 1865. A treatise by Chvostek ("Das einfache Duodenal Geschwür," "Wien. med. Jahresbücher," 1883) presents the literature up to 1882. From this year to 1891 the literature is contained in a dissertation by Oppenheimer ("Das Ulcus Pepticum Duodenale," Inaug. Diss., Wurtsburg, 1891). But perhaps the most complete compilation of the literature, bringing it up to the year 1900, is found in the masterly treatise on "Ulcer of the Stomach and Duodenum," by Samuel Fenwick and W. Soltau Fenwick (P. Blakiston's Son & Co., Philadelphia, 1900). The surgical side of duodenal ulcer and its corresponding literature were ably represented by an article on "Perforating Duodenal Ulcer" by Dr. Robert F. Weir.*

ULCERATIONS IN CONSEQUENCE OF NECROBIOTIC PROCESSES.

Peptic Duodenal Ulcer—Round Duodenal Ulcer.

Existence, Concept, and Etiology.—Duodenal ulcers are observed much more rarely than gastric ulcers, although their origin and cause are the same. Their form is that of a round, sharply defined, funnel-shaped excavation, the edges of which descend in terraces. The floor of the ulcer is usually free from necrotic tissue and pus. The duodenal ulcer varies in size between that of a lentil and a silver dollar. It is *almost always found on the upper horizontal branch of the duodenum*, between the pylorus and the mouth of the common gall- and pancreatic duct, and only *exceptionally* on the *lower section of the duodenum*. If it is very close to the pylorus, it usually passes over to the mucous membrane of the stomach also.

---

* "President's Address in Surgery," delivered before the American Surgical Association, "New York Medical Record," vol. LVII, No. 18, May 5, 1900, p. 749.

Location.—According to Morot (*l. c.*) and Vonwyl (*l. c.*), ulcers of the duodenum are situated between one and one and one-half inches of the pylorus, but Schwartz ("Ulcère perforante du duodenum," "Bullet. et Mém. Soc. de Chirurg.," January, 1898) gives one case in which the perforation existed on a level with or a little below the ampulla of Vater, and thus allowed a free escape of bile into the peritoneal cavity. The figures given by recent authorities show that in 262 cases of Collin (*l. c.*) the perforation occurred 242 times within two inches of the pylorus, in the descending portion 14 times, in front of the aorta 3 times, and in the ascending portion 3 times; or, expressing the same a little differently, 162 of the 262 cases were situated at from two to four millimeters from the pylorus; 75 close to it; 4 encroached on the pylorus itself; and 2 extended through the pylorus to the duodenum and to the stomach. Perry and Shaw (*l. c.*) found, in 141 cases of duodenal ulcer, 123 in the first part of the duodenum, 16 in the second part, and only 2 in the third part. In most cases only one ulcer is found; in rare instances two, three, or even four were observed.

Sex.—Contrary to the peptic gastric ulcer, the duodenal ulcer occurs more frequently in men than in women—the proportion is about three to one. About 79 per cent. occur in males according to Collin. From tables of Morot, Chvostek, Kraus, and Lebert 176 cases of duodenal ulcers were collected by Weir, and of these, 144 were in men and 30 in women.

The greater frequency of duodenal ulcer in the male sex appears paradoxical when considered from the standpoint of the comparative immunity from gastric ulcer which the male enjoys. This predisposition to duodenal ulcers in the male is explained by Boas by the different habits of life that distinguish the male from the female sex, especially the excessive use of tobacco and alcohol, which induces chronic forms of gastritis acida that are accompanied by excessive secrection of HCl. As this is not sufficient to explain why the duodenum should be the most frequent site of the peptic ulcer, he cites the experiments of Moritz, which show that the stomach first expels the more liquid parts of the chyme, and gradually permits the more consistent and solid portions to follow later on (Moritz, "Verhandlungen der LXV. Versammlung der Gesellschaft deutscher Naturförscher u. Aerzte in Nürnberg," 1893). From the investigations of von Mering also we know that the stomach expels water into the duodenum in an exceptionally short time. It is true that alcohol, salts, peptones, dextrin, and acid are absorbed from the stomach, but these experiments demonstrate that they are absorbed in very small quantities. Von Mering's experiments show, furthermore ("Verhand. d. Cong. f. innere Med.," 1893), that the first quantities of liquid expelled from the stomach contain these dissolved substances in a very concentrated state. If, for instance, he poured in 300 c.c. of a 25 per cent. solution of alcohol, 150 c.c., containing 10.5 per cent. of alcohol, flowed out into the duodenum within ten minutes. Similarly, if he poured 200 c.c. of a 50 per cent. solution of glucose into the empty stomach, the amount of liquid escaping into the duodenum in ten minutes amounted to 120 c.c., containing 32 per cent. of sugar. All this goes to show that the stomach thus possesses a means of protecting itself against high concentrations by the expulsion of liquid; but the duodenum does not possess such a self-protective function: it necessarily is more liable to be irritated by concentrated substances soluble in water than the stomach, which, in addition to this, is protected by the larger quantity of semisolid chyme that is capable of absorbing these soluble irritants and keeping them from the mucosa. Boas (*l. c.*, p. 297), Boucquoy ("Arch. gén. de Méd.," 1887), and Burwinkel (*l. c.*) have made the observation that duodenal ulcer is a frequent occurrence in habitual alcoholic subjects.

Gastric ulcer, as has been described in the author's work on "Diseases of the Stomach," is caused by the action of the HCl and pepsin of the gastric juice on those parts of the mucous membrane of the stomach the nutrition of which has been lessened by disturbances in the circulation, and the power of resistance correspondingly reduced. The occurrence of peptic duodenal ulcers is to be attributed to the same causes. When the contents of the stomach, containing an excess of free HCl and pepsin, especially in the second period of the gastric digestion, pass into the duodenum, the HCl is gradually neutralized by the addition of bile and alkaline pancreatic and intestinal juice, while the chyme is being carried downward. By this the digestive power of the pepsin is destroyed. We can, therefore, easily understand why the duodenal ulcer is usually located in the uppermost part of the duodenum. If the neutralization of the HCl, from any reason at all, requires more time than usual, so that the chyme has not entirely lost its digestive power, even in the lower part of the duodenum and in the first part of the jejunum, peptic ulcers may also, under certain conditions, be formed in these localities. Since the self-digestion of the duodenal mucous membrane under the influence of HCl and pepsin takes place only when its nourishment is lessened at some particular points, *disturbances in the circulation are always essential etiological conditions* for the development of a duodenal ulcer. These disturbances may be caused by arteriosclerosis, embolus of the arteries, thrombosis of the veins, venous hyperemia resulting from affections of the heart, the lung, and the liver, compression or thrombosis of the portal vein, losses of blood after trauma, the caustic effect of toxic substances, and from many other causes. Emboli of single branches of the duodenal arteries and also embolic ulcers which may rapidly increase in size under the effect of the HCl and pepsin have often been observed, not only in adults, but also in children, and especially in new-born babes. The so-called melæna neonatorum may be traced in some instances, according to Merkel, Nothnagel, and Pavensky, back to embolic processes. It has not yet been definitely settled whether the duodenal ulcers of septicemia are due to a hemorrhagic inflammation of the mucous membrane or to emboli. The same doubt exists regarding the intestinal ulcers observed in association with erysipelas and pemphigus (Larcher, Malherbe). It may be asked why so much importance is attributed to pepsin and HCl as an etiological factor and not to the digestive ferments of the pancreatic juice. The reason of this is found in the fact that the peptic ulcer is found only in localities which can be reached by the gastric secretion—namely, the esophagus, stomach, and upper duodenum. If the pancreatic juice were an active factor, we should find them only in the duodenum. We know that pepsin and trypsin digestion, though affecting proteids, practically act in a different manner (Ad. Schmidt, *l. c.*). It may be that pepsin more readily digests the epithelial cells than trypsin.

Age.—No age is exempt from this disease. Born and Spiegelberg incline to the opinion that duodenal ulcer may develop during intra-uterine life. Chvostek (*l. c.*), in 87 autopsies on children under ten years of age, found five duodenal ulcers, of which one was seen in a child seven weeks old, another in a child four days old, and a third in a babe of three hours. Collin's table is the most complete in this respect, and it is here presented. In 297 cases of duodenal ulcers they were met with:

Under 10 years of age,	42	times.
Between 10 and 20,	24	"
" 20 " 30,	43	"
" 30 " 40,	52	"
" 40 " 50,	46	"
" 50 " 60,	41	"
" 60 " 80,	28	"
" 81 " 95,	3	"

Duodenal ulcers are found most frequently in persons between thirty-one and sixty years of age. They are also found fairly often in children under ten years of age, as well as in newly born children, whereas gastric ulcer at that age is extremely rare.

Pathologic Anatomy.—The anatomical configuration of the duodenal ulcer is, in general, the same as that of the gastric ulcer, a description of which, given in the author's work on "Diseases of the Stomach," will make a second detailed account unnecessary. In most cases it is located at the uppermost part of the duodenum, above the mouth of the common gall-duct and pancreatic duct, and only exceptionally below the same. It is found very often in the immediate neighborhood of the pylorus, and then shows a preference for the rear duodenal wall. In the descending part it is found very often on the inner wall, and more especially when it is near the duodenal papilla. If the ulcer has attained considerable size and depth, the formation of a duodenal stricture gradually ensues as a result of a contraction in the cicatricial tissue. This stricture first causes an expansion of those parts of the duodenum lying above it; occasionally also the formation of large-sized diverticula, and eventually also dilation of the stomach and an enlargement of the lower section of the esophagus may result. If the duodenal ulcer is located at the mouth of the common gall-duct, the latter is closed by the cicatrization, and permanent jaundice may set in. If the ulcerative process descends downward very quickly to the serosa, larger vessels of the intestinal walls are often eroded, and losses of blood in varying quantities occur, which occasionally may cause death in a short period of time. A rupture into the portal vein, and eventually into the aorta, was observed by Grünfeld and Stich as the result of a healed duodenal ulcer, and Frerichs observed a compression of the portal vein and resulting thrombosis as a result of the same cause. Ichorous and extended suppuration, which may extend along the larger vessels and come to the surface on the back or in the region of the neck, may set in when the duodenal ulcer breaks through into the retroperitoneal region. If the duodenal ulcer extends quickly to the serosa, perforation peritonitis results. If, however, this be prevented by a prior agglutination of the duodenum with neighboring organs, and the ulcerative process extends to these also, abnormal communications with other intestinal loops or with the gall-bladder may be formed. If the break is into the liver or the pancreas, extended suppuration and destruction of these organs take place. Just as in the case of gastric ulcer, the formation of pyopneumothorax or subphrenic abscess ("pyopneumothorax subphrenicus") has been observed in conjunction with a duodenal ulcer by von Leyden. In this case a cavity filled with pus alone—pyothorax—or with pus and air—pyopneumothorax—arises between the liver, the diaphragm, and the stomach, which forces the diaphragm upward and the liver downward, and thus may simulate a pyopneumothorax. It is very rare that the duodenal ulcer breaks through the anterior abdominal wall after the agglutination of the same with the duodenum, but external intestinal fistulæ have been described as a result of such perforation.

In two cases observed by Eichhorst and Ewald a cancer developed on the base of a duodenal ulcer, whereas, according to the investigations of Hauser, this is observed much more frequently in the case of gastric ulcer.

Symptomatology.—In a part of the cases (20 per cent., Morot) the duodenal ulcer remains for a long time without any symptoms, and thus is latent until a considerable loss of blood calls attention to the disease. An open or a completely healed ulcer is often found at the autopsy which during life caused no symptoms, or only such slight ones that they were not noticed. These ulcers are occasionally not recognized until at autopsy they are discovered to have been causes of fatal hemorrhage or perforation peritonitis.

Pain.—In a great many cases the duodenal ulcer causes subjective complaints, and more especially pains of greater or less intensity. These pains, however, are so similar to those observed in gastric ulcer that a positive differentiation between the two is impossible, especially if the duodenal ulcer is situated near the pylorus. To be sure, it is stated in many text-books that the pain in the case of duodenal ulcer is confined to the duodenal region,—lower edge of the liver in the parasternal line, to the right of the pylorus and somewhat below it,—and that the sensibility of pressure is also most pronounced at the same place. The same is true, however, in the case of a gastric ulcer which is situated in the region of the pylorus. Besides, the pains in the case of duodenal ulcer very often radiate toward the epigastrium, and it is never possible to ascertain, with any degree of certainty, where the sensibility to pressure and the pains are greatest, on account of the defective power of localization in most patients. Some clinicians (Kreuz) attach great importance to the fact that the pains in the case of duodenal ulcer radiate upward—into the epigastrium and the upper abdominal region, and also downward toward the pelvic region, but not toward the loins. This, however, is occasionally observed in gastric ulcer, and especially in pyloric ulcer. It is also often given as an important means of differentiating these two diseases that in the case of duodenal ulcer pain sets in, as a rule, later after a meal—from one-half to three hours later—than in the case of gastric ulcer, where it sets in almost immediately. Experience teaches us, however, that even in gastric ulcer the pains may appear a longer time after eating and reach their greatest intensity when the digestion is at its height and when the contents of the stomach contain the greatest amount of free HCl, the ulcer being very much more irritated by the free acid. The early or late commencement of the pains is also dependent to a considerable extent upon the amount and kind of food introduced in the stomach. That the pains in the case of duodenal ulcer are often most intense several hours after the meal may very probably be traced to the fact that at this time the chyme contains large amounts of free HCl, and is carried more quickly into the duodenum than during the first period of gastric digestion.

It has been stated by Chvostek that pains, which in the case of duodenal ulcer set in two and a half hours after breakfast and three hours after dinner, may be prevented or at least alleviated by the use of wine, while in the case of gastric ulcer its use increases the pain. He explains this observation by assuming that the irritation of the alcohol causes a closing of the pylorus lasting for some time, so that in consequence of this no chyme can pass over into the duodenum for a longer period of time and irritate the ulcer. In my experience with the gastrograph, however, it was evident that wine or alcohol did not have this effect, for in numerous observations the contents of the stomach passed over into the intestines much more quickly after a moderate consumption of alcohol than without it. Alcohol is absorbed fairly rapidly from the stomach, the contents losing its percentage of this substance. It is much more reasonable to assume that the contents of the stomach are very much diluted by the water in the wine, while the alcohol may irritate more strongly any gastric ulcer which is present before it is absorbed. Excessive amounts of alcohol—for instance, 20 per cent. of the gastric contents—lead to a retardation of gastric peristalsis (see Hemmeter, "Diseases of the Stomach," second edition, p. 294).

The character of the pains in the case of duodenal ulcer is the same as in the case of gastric ulcer, although they are usually frequent and severe in the case of the former than in the case of an ulcer of the stomach. If the duodenal ulcer is advancing or if the ulcerative process has extended to the serosa, pains may set in during the night and also in the morning on an empty stomach. These pains, as a rule, however, increase during the intestinal digestion.

Vomiting.—In the clinical histories of the authors thus far cited the objective symptom of vomiting is very rarely spoken of. Krauss states that in the few cases that he came across a reference to the vomiting, it either depended on strictures of the duodenum or it constituted the culminating point of an attack of gastralgia. Oppenheimer mentions vomiting only 17 times in over 100 cases. Similarly, the reports of Elbers ("Darmgeschwüre," 1831), Boucquoy (*l. c.*), Starke ("Deutsch. Klinik," 1870), Mayer ("Die Krankheiten des Zwölffinger Darmes," Düsseldorf, 1844), and S. and W. S. Fenwick (*l. c.*) indicate the rather small percentage of attacks of vomiting in cases of duodenal ulcer. In the reports of 260 cases of duodenal ulcer which I found in the literature there was an account of vomiting in 20 per cent. of the cases. Reckmann ("Ulcus Duo., u.s.w., Diagnose," Thesis, Berlin, 1893) speaks of three types of vomiting occurring at three different periods, the first being insipid and watery, the second bitter and thick as syrup (bile), and the third being a sweetish liquid (blood). He does not precisely state when and under which conditions these three kinds of emesis occur.

Hematemesis.—If, in consequence of the erosion of larger blood-vessels, a great loss of blood has taken place, the blood is usually discharged both by vomiting and by evacuation. More rarely the latter alone is observed. It is well known that this is also true in the case of gastric ulcer. If the loss of blood is very great, death may ensue before all the blood was discharged by means of evacuation and vomiting. If the duodenal ulcer breaks through extraperitoneally into the retroperitoneal connective tissue, extensive suppurations, even up to most distant parts of the body, set in. These suppurations, even when they cause fever, are then discovered only when the pus is discharged externally. If the break is intraperitoneal, before the duodenum has been agglutinated with its neighboring organs, it causes perforation peritonitis. In this case the pain at first is most severe at the place of rupture, and radiates thence toward the upper and lower abdominal regions. Later on, however, the pains are of quite diffuse nature.

Small losses of blood are much more likely to be overlooked when they occur in the intestinal evacuation than if they appear in the vomit. Many patients have been brought dangerously near death from intestinal hemorrhages without having been in the least aware that they were passing blood in their evacuations. This fact must be borne in mind in estimating the diagnostic value of the statistics on these symptoms. According to Chvostek, Krauss, and

Oppenheimer, about one-third of all cases of duodenal ulcer are accompanied by loss of blood in the intestinal evacuations or in the vomited matter. In 34 cases observed by Oppenheimer there were 16 in which the blood was evacuated both ways. In 10 it found its way out by the stool only, and in 8 by the vomit only. Death from loss of blood may occur without either melena or hematemesis. Intestinal hemorrhages may occur at regular intervals, and be preceded by pain and dyspeptic distress at each of these intervals. In a case referred to by Weir (*l. c.*) the question of the presence of a duodenal ulcer in a patient with intestinal hemorrhage, right-sided epigastric pain, and distress some time after eating was decided negatively because blood was discharged per anum, of a bright red color and unclotted. The patient, moreover, had hemorrhoids, the presence of which led to the confusion in diagnosis. Collin gives twelve instances in which the blood came from the pancreatico-duodenal artery; three times the gastro-epiploica dextra was opened into; twice the pancreatic artery; once the hepatic; twice the aorta; twice the vena porta; and once the superior mesenteric vein. Pagenstecher states that the ulcers on the posterior wall are more apt to bring about hemorrhage than the anterior ones, as the vessels are more abundant and larger in that direction.

Frequency.—Perry and Shaw (*l. c.*), in an analysis of the reports of 13,055 autopsies occurring at Guy's Hospital from 1826 to 1892, in which the condition of the duodenum was noted, found an open ulcer of the duodenum in 34 cases, or 0.26 per cent. Ulcer of the duodenum heals with greater difficulty and with less frequency than ulcer of the stomach, and the two Fenwicks are of the opinion that chronic ulcer of the stomach is about ten times as common as the same ulcer of the duodenum. According to their statistics, only 1.7 per cent. of cases of gastric ulcer are accompanied by a similar condition in the duodenum. Duodenal ulcer is, accordingly, a rare lesion. Collin ("L'ulcère simple du duodenum," "Thèse de Paris") could find only 26 cases in a range of ten years. Houze ("De l'ulcère perforat du duodenum," "Thèse de Paris," 1896), in three additional years, could add only 3 more cases, though Darras ("De la perforation de l'ulcère simple du duodenum," "Thèse de Paris," 1896), more than one year later, found 5 others. Since attention has been given to the subject, and particularly to the surgical aspect of it, by Robert F. Weir in 1896, when 9 cases were reported as having undergone surgical treatment, there have been reported 51 operations for this disease.

The relative frequency as a cause of death, deduced from autopsies, is as follows:

Willigt,	1,600	sections with	74	gastric ulcers and	2	duodenal.
Starke,	384	" "	36	" " "	3	"
Crinfeld,	1,150	" "	124	" " "	4	"
Steine,	3,085	" "	89	" " "	12	"
Vonwyl,	12,806	" "	98	" " "	3	"
Latham,	8,192	" "	. .	" " "	12	"
Perry and Shaw,	17,652	" "	. .	" " "	70	"
New York Hospital (unpublished),	1,000	" "	. .	" " "	2	"
	45,869		421		108	

In about 0.2 per cent. of these cases duodenal ulcer was assigned as the cause of death.

Kinnicutt (Jacobi, "Festschrift," 1900), however, in 30,000 autopsies, which probably included many embraced in the above table, has given a rather higher percentage of frequency, placing it at 0.4 per cent. Though the proportion is a small one, stress must be laid on the fact that from 40 to 70 per cent. of these ulcers perforate, with an almost invariable fatal result. The relative proportion of gastric to duodenal ulcers is 12 to 1 (Burwinkel).

Frequency of Perforation.—Perforation is said to have taken place 181 times in Collin's 262 cases, or in 69 per cent.; Chvostek, however, places the frequency of perforation at about 42 per cent. of the cases. In these 262 cases of Collin there was a generally diffused and fatal peritonitis 125 times. But occasionally in anterior perforations, but more commonly with the perforations existing elsewhere, adhesions or ulcerations with abscess and hemorrhage from the duodenal wall or adjacent organs have been encountered. Examples of such complicating lesions are subphrenic abscess, which, according to Oppenheimer, follows more commonly a perforation in the superior wall of the horizontal portion of the duodenum. Hepatic and pancreatic adhesions are at times met with—Oppenheimer gives 6 cases and Collin 38 cases of such adhesions. Perforations into the gall-bladder have been reported, but more important and dangerous are the ulcerations into the aorta (Stich, 2 cases, "Deutsch.

Arch. f. klin. Med.," 1874), the vena porta (Rayer and Habershon, 2 cases), the superior mesenteric vein (Warfinger), or the hepatic artery (Vonwyl). The ulceration has also been known to invade the adjacent colon (Murchison and Saunderson, 2 cases, quoted from Robert F. Weir, "New York Medical Record," May 5, 1900).

Perforation peritonitis is observed much more frequently as a result of duodenal ulcer than in the case of gastric ulcer, because the walls of the duodenum are comparatively thin. It causes death usually in from twelve to thirty hours, with the symptoms of heart failure and collapse. If, in consequence of a perforation, "pyopneumothorax subphrenicus" sets in, the patients complain of precordial fear, great dyspnea, and a collection of liquid and air may be discovered by percussion and auscultation in the upper hepatic region. If the duodenal ulcer has grown together with neighboring organs, especially with the pancreas, or if a cancer has developed in it, a distinct resistance is sometimes palpable at the lower edge of the liver in the duodenal region. If the duodenal ulcer is near the mouth of the common gall-duct (according to Fleischer, 6 per cent. of the cases), permanent jaundice almost invariably ensues.

Disturbances on the part of the stomach—gastralgia, nausea, loss of appetite, vomiting—are, as a rule, much less pronounced in connection with duodenal ulcer than in the case of gastric ulcer. If, as the result of cicatrization of a large duodenal ulcer, a duodenal stricture occurs, the symptoms of a dilation of the stomach are observed if the stricture has reached an advanced stage. The anatomical stricture of the duodenal peptic ulcer does not differ essentially from the gastric lesion of the same type. The general condition and the nutrition of those suffering with duodenal ulcer are at first but little affected. If, however, the patients take little food on account of violent pains, and if the erosion of the blood-vessels causes repeated losses of blood, the patients become anemic, emaciated, and finally succumb to heart failure.

Latency.—The frequency with which acute and chronic duodenal ulcers may run their course without giving rise to symptoms is demonstrated by the statistics of Perry and Shaw ("Guy's Hospital Reports," 1893, p. 171). Out of 151 cases collected by these observers, 91, or 60 per cent., gave no clinical symptoms of importance prior to the termination of the cases by hemorrhage or perforation. In 20 out of 25 instances of perforation the patients were so slightly indisposed prior to this occurrence that they had not consulted a physician (Schwartz); only 5 of these cases had previous gastric symptoms. Collin states that 53 per cent. of the cases analyzed by him remained latent during their entire course ("Scot. Med. and Surg. Journal," July, 1897). Samuel and W. Soltau Fenwick (*l. c.*) state that only 32 out of 43 cases, or 74 per cent., had suffered from abdominal pain, vomiting, or hemorrhage some time before death.

Diagnosis.—It may be seen, from the description of the symptoms, that if we exclude the cases where duodenal ulcers are caused by extensive skin burns, septicemia, erysipelas, pemphigus, and amyloid degeneration of the intestines, a positive differentiation of gastric ulcer, especially if it is located in the immediate neighborhood of the pylorus, from the duodenal ulcer can not be made during life. The diagnosis is one of probability. We can suspect a duodenal ulcer only when we have to do with strong, healthy men or with children under ten years of age, because in the latter it is quite the exception to find gastric ulcers. We suspect it in these cases if the pains and the sensibility to pressure are greatest in the duodenal region; if the pains radiate only upward and downward, but not toward the loins, and always set in some time after the meal; if, in the case of hemorrhages, the blood is carried off only in the evacuations; and if the stomach complaints are but slight. The diagnosis is strengthened by the occurrence of permanent jaundice (seat of the duodenal ulcer, in the neighborhood of the duodenal papilla) and perforation peritonitis, which is rarer as a result of gastric ulcer. The diagnosis of gastric ulcer may be made with probability if the following objective signs and symptoms are present: (1) The signs of gastric ulcer appear in anemic girls suffering with chlorosis; (2) the pains and sensibility to pressure are confined to the epigastrium; (3) the pains are felt immediately after eating, growing more intense when digestion is at its height—*i. e.*, simultaneous, greatest increase in the free HCl of the contents of the stomach; (4) they are only transiently alleviated by the introduction of milk or of sodium bicarbonate; (5) other disturbances on the part of the stomach grow more prominent; (6) superacidity can be proved for the gastric juice; and (7) the symptoms of dilation of the stomach are lacking (seat of the ulcer, on the front or rear wall, but not in the pylorus).

The number of quantitative determinations of free HCl in the gastric contents after test-meals is as yet too small to permit of accurate conclusions. In three cases of duodenal ulcer, proved by autopsy, in which I made such analyses the amount of free HCl was normal in two cases and excessive in one. The figures, expressed in terms of $\frac{1}{10}$ normal solution of NaOH, were 28°, 26°, and 38° respectively. It has been urged that hyperacidity is not always present in gastric ulcer, and therefore could not be used as a distinguishing sign. There are, it is true, very rare cases of peptic ulcer of the stomach with normal or even low amounts of free HCl, but they are so exceptional as to be disregarded in a consideration of diagnostic factors. This low or normal acidity may be due to an exhaustion of the oxyntic cells as a result of prolonged overwork.

Vonwyl has grouped the distinguishing diagnostic factors in gastric and duodenal ulcers in the following schema:

Gastric ulcer: (1) More frequent in women from the twenty-fifth to the fiftieth year. (2) Pain promptly after eating. (3) Relieved by vomiting. (4) Frequent vomiting of bile, mucus, and food. (5) Marked dyspeptic symptoms. (6) Frequent bloody vomiting. (7) More seldom, bloody stools.

Duodenal ulcer: (1) Occurs most frequently in males. (2) Pain in right hypochondrium or to right of parasternal line. (3) Comes on in from two to four hours after meals. (4) No relief by vomiting; latter not frequent. (5) Bloody stools (melena or bright blood) more common than bloody vomiting. (6) If jaundice is present, this would contribute to the diagnosis.

Considering the value of the clinical features from a diagnostic standpoint, so far as I am able to judge from personal experience, and adding to this the experience of the most reliable native and foreign clinicians of eminence, the conclusions seem to be inevitable that the diagnosis of duodenal peptic ulcer will rarely be made, and if it is made correctly and proved as such, it is due to an exceptional combination of very convincing and unmistakable signs and symptoms or to an accident. The main factors upon which one has hitherto placed dependence in the diagnosis were the pains, the vomiting, the hematemesis and melena, and the icterus occurring in association with these. I have already critically considered the diagnostic value of the pains as symptoms. It has been said that the pain of duodenal ulcer is situated in the right hypochondrium, as a rule; but this may also be the case with gastric ulcer. On theoretical grounds it has been stated that the pains due to a duodenal ulcer occur only when the gastric chyme is expelled into the duodenum—that is, in from three to six hours after the ingestion of the food; whereas the pain of gastric ulcer occurs immediately on the ingestion of food. Furthermore, it was asserted that the pain of gastric ulcer is relieved by vomiting, but that due to duodenal ulcer persisted after emesis. All these signs are unreliable, for the pain of duodenal ulcer, even assuming that it is always due to the entrance of the gastric chyme, depends upon the rate of the gastric peristalsis, which may vary greatly. In one of my cases of duodenal ulcer, proved as such at the autopsy, the pain occurred regularly within from twenty to thirty minutes after the ingestion of food; and in the majority of reports in which any statement is ventured concerning the time elapsing between ingestion of food and the pain of duodenal ulcer it is stated as varying between two and three hours. The pain of gastric ulcer is not always relieved by vomiting. Many times it persists for a long period after emesis has stopped. No information of diagnostic value can be gained from the dyspeptic symptoms, the appetite, and the condition of the stool, which may be diarrheic or constipated. The results of test-meal analyses in duodenal ulcer are varying. Such have been carried out by Devic and Roux ("Province Medicale"), A. Robin (cited by Collin, *l. c.*), Reckmann (*l. c.*), and von Leube ("Special Diagnosis of Internal Diseases"). Boas, Devic, Roux, and Robin found hyperchlorhydria; Leube and Reckmann found subacidity. As already stated, I have found normal acidity in two cases, and excessive acidity, or hyperchlorhydria, in one case. Robin found absolute anacidity and considerable quantities of organic acids in three cases. It is conceivable that hyperacidity of long standing may cause exhaustion of the oxyntic cells and eventuate in total anacidity from overwork of the secretory apparatus. Concerning the frequency of the hemorrhages, I have already spoken. No diagnostic information can be gained from the analysis of the urine in these cases. The icterus, to which much diagnostic importance is attributed by a number of our writers, is not due to the ulcer itself, but rather to its complications—viz., stenosis of the duodenum and cicatrization near the ampulla of Vater. The most useful diagnostic elements, though they are not absolutely constant, are: (1) The prevalence of duodenal ulcer among men; (2) hematemesis and melena, but especially melena without hematemesis; (3) the absence of the dorsal pain-point of gastric ulcer; (4) a painful area in the right hypochondrium or umbilical region, rather than in the epigastric region. Burwinkel states that intestinal hemorrhage in large amounts is very rare, excepting enteric fever and vicarious menstruation, and should always excite the suspicion of gastric or duodenal ulcer. He asserts that a differential diagnosis between gastric and duodenal ulcer is possible, and that by careful study of the symptoms he has been able to diagnose five cases of duodenal ulcer in the last few years. Ewald, Penzoldt, Nothnagel, and Lemke hold that the diagnosis between gastric and duodenal ulcer is very difficult and practically impossible.

Use of the Stomach-tube for Diagnostic Purposes.—There can be no doubt that the employment of a stomach-tube in gastric and duodenal ulcer is associated with danger. Grave, even fatal, hemorrhages have frequently been reported as immediately following upon lavage of the stomach, and the danger of a perforation is not to be overlooked. Twenty years ago the use of a hard, stiff sound and the old-time stomach-pump was advised by Schliep ("Deutsch. Arch. f. klin. Med.," Bd. XIII, S. 456). The introduction of the soft stomach-tube has lessened these dangers somewhat, but then it can not be denied that the evil results following the employment of the tube in some cases are not due to the instrument itself directly, but to the convulsive movements caused by the nausea and vomiting. If the tube can in any particular case be introduced without causing nausea and vomiting, or if this nausea can be prevented by the application of cocain to the throat, the dangers referred to are very much lessened. Passing a stomach-tube is contraindicated in the following conditions: (1) In all acute ulcers showing (2) a disposition to hemorrhage, as evidenced by the hematemesis; (3) if the symptoms and signs of ulcer have only existed for a short time; (4) in the three or four weeks following a hemorrhage, in which period the classical treatment for ulcer should have been carried out; (5) when there are indications that the ulcer is situated in the esophagus or cardia; (6) when the sensitiveness to pressure is very great in the epigastrium and in the right hypochondrium. But in chronic ulcers of an evidently torpid character, with no disposition to hemorrhage or perforation, the use of the stomach-tube for therapeutic measures is permissible in such cases as have resisted dietetic and other treatment. Medicated lavage of the stomach under such conditions greatly favors cicatrization. By the same means the hyperacidity, which is a causative factor, may be very much reduced. Whenever, however, such treatment has to be undertaken in cases of suspected duodenal ulcer,—for even here the gastric hyperacidity is a recognized cause, and therapeutic control of this hyperacidity has, in my opinion, occasionally led to cure of the duodenal ulcer,—it should be undertaken only after instructing the patient and the relatives of the possible dangers of the method. The first lavage under such conditions had best be undertaken only in consultation with another physician. The tube should be introduced very gradually, after anesthetizing the throat with a 4 per cent. solution of cocain. Should a slight hemorrhage occur, it should be treated according to the principles laid down in the chapter on "Hemorrhage from the Stomach" in the author's work on "Diseases of the Stomach." Far from being absolutely contraindicated in the treatment of gastric and duodenal ulcer, Ewald actually recommends irrigation of the gastric mucosa with iced water for the arrest of hemorrhage into the stomach ("Klin. d. Verdauungs-Krankheiten," Berlin, 1893). In any case, if the patient has stood the lavage for five days without any signs of irritation or of blood, the danger of hemorrhage becomes less with every day, and the ulcer, whether gastric or duodenal, can be treated more effectively in this manner. The use of the stomach-tube for diagnostic purposes in gastric ulcer should be controlled by the same principles as are laid down in the preceding. I come now to the consideration of a method by which the diagnosis between gastric and duodenal ulcer may be made with some certainty. This method involves the use of the gastroscope.

Gastroscope.—In cases that have become first habituated to the stomach-tube and in whom such symptoms are presented as make the diagnosis between gastric and duodenal ulcer unsettled, direct inspection of the stomach will, under favorable conditions, permit of the exclusion of gastric ulcer, since those portions of the stomach which are most frequent sites of ulcer are capable of being inspected with the gastroscope. It is suggested, therefore, that in cases in which the symptoms of hemorrhage from the stomach or intestines are presented, together with pain in the epigastric, umbilical, or right hypochondriac region, especially if the blood appears only in the stools, a gastroscopic examination of the stomach be made by a specialist experienced in the manipulation of the instrument after the patient has become habituated to the stomach-tube. This procedure appears to me to be the only way of arriving at a clear diagnosis, and as life depends upon this diagnosis, the method is justifiable if executed by trained hands. The following tables illustrate the relative frequency of correct and incorrect diagnosis in duodenal ulcer.

Weir (*l. c.*) states that it has been possible in thirteen of the more recent instances either to arrive at a correct preoperative diagnosis or to make the alternative diagnosis, varying between a duodenal and a gastric perforation. The difficulties that have been experienced are succinctly shown in the following statement:

Diagnosis wrong—ulcer not found:	
Diagnosis as acute peritonitis,	8 times
" " appendicitis,	15 "
" " intestinal obstruction or appendicitis,	1 "
" " strangulated hernia,	1 "
" " hydronephrosis,	1 "
	26
Diagnosis wrong—ulcer found:	
Diagnosis as acute peritonitis,	5 times
" " intestinal obstruction,	4 "
" " appendicitis,	3 "
	12
Diagnosis right or approximately so—ulcer found:	
Diagnosis as duodenal ulcer,	5 times
" " gastric or duodenal ulcer,	6 "
" " appendicitis or duodenal ulcer,	2 "
	13

Diagnosis of Perforation of Duodenal Ulcer.—The duodenum in its first part is free and covered by peritoneum. I have already spoken of the most frequent site of the perforation, which is encountered on the anterior wall; but perforation with extravasation is also possible posteriorly, in the first part of the duodenum. In this case the extravasation is more likely to enter the omental bursa (the lesser omental cavity) or invade the retroperitoneal tissue. The anterior perforations are, for obvious anatomical reasons, less likely to be closed off by adhesions, and hence a prompt and profuse involvement of the peritoneum takes place from the opening there located. The frequency of the perforation has also been referred to. If this accident occurs when the stomach is filled, the extravasation usually follows an anatomical course, which has been outlined by Pagenstecher ("Die chir. Behandl. des Duodenalgeschwürs," "Deutsch. Zeitschr. f. Chir.," August, 1899) and been confirmed by Weir in an experience of three perforations of the gall-bladder. Fluids escaping from the duodenum or gall-passages accordingly flow at first between the liver and the colon, thence along the ascending colon, sometimes to the outside of the bowel, and sometimes between the omentum and the parietal peritoneum toward the iliac fossa. From this point they may pass over to the sigmoid flexure or into the pelvis.

Signs and Symptoms of Perforation; Results of Operations.—The symptoms of shock and pain are necessarily severe. In 26 out of 47 of Weir's cases great pain was felt in the epigastrium or in the right of this region. In the remaining 21 cases of these 47 it was similar [illegible] but it has been observed a few times [illegible] a frequent sign [illegible] Vomiting is reported [illegible] out of 47 instances. The peritoneal symptoms [illegible] rapidly sometimes exhibit a tendency [illegible] epigastric and hypochondriac regions. Should the peritonitis [illegible] at the site of the umbilicus or below it, the mistaken diagnosis of appendicitis is very likely to be made. The disappearance of the liver dullness, as in gastric ulcer, is suggestive of the escape of air through a perforation in the gastro-intestinal tract. As the only chance of recovery from a duodenal perforation depends upon a successful operation and closure of the opening, the earlier the diagnosis of perforation is made, the greater the probability of saving life. Mayo ("Surgery of the Stomach," "Lancet," March 10, 1900) states that of 429 cases of gastric perforation, 82 were operated on in the first twenty-four hours. Of these, 47 recovered and 35 died—42 per cent.; but in operations done after that period, over 70 per cent. of the patients died. The same observations can be made with regard to the success of operations for duodenal perforations. In the 51 cases collected by Robert F. Weir (*l. c.*) the lesion was recognized and closed at operation in 25 cases; of these, 13 underwent operation after thirty hours' delay, and all resulted fatally; 12 other patients were operated on within that time, and 8 survived, giving thus only 33⅓ per cent. of mortality. This marks an improvement on the statistics of Pagenstecher, which gave a mortality of 60 per cent. in the found and sewn-up duodenal ulcers located by him. An unmistakable advance in the diagnostic ability is shown in the following statistics:

Prior to 1895, 28 cases reported in which laparotomy was done for duodenal perforation: in 15 (75 per cent.) ulcer not found; in 5 (25 per cent.) ulcer found and sutured; in only 2 cases (10 per cent.) was the possibility of duodenal perforation considered.*

Since 1895, 31 cases operated: perforation not found in 11 per cent.; perforation found and sutured in 20 cases (65 per cent.); its presence was considered in diagnosis in 11 cases (35 per cent.).

Progress.—In the statistics on the frequency of perforation is also given the percentage of duodenal ulcers which run their course without perforation. Collin gives an account of 39 cases of cicatrices from ulcerations in the duodenum. Perry and Shaw (*l. c.*) found unmistakable evidences of repair in nearly 50 per cent. of their cases. Such healed duodenal ulcers may prove harmless, and the duodenum become restored to integrity, but they may also cause dangerous narrowing of the lumen of the duodenum; the cicatrix may involve the outlets of the pancreatic and bile-ducts; it may so distort the anatomical course of the duodenum as to obstruct the outlet of the pylorus and also of the bile-ducts, causing dilation of the biliary passages and of the stomach.

Prognosis.—In the case of duodenal ulcer the prognosis is much more unfavorable than in gastric ulcer. This is because in duodenal ulcer peritonitis causes death more frequently, and because losses of blood often cause advanced anemia, and also because in the case of larger ulcers the formation of a duodenal obstruction is almost inevitable.

---

* The foregoing statistics quoted from Weir probably contain a numerical error, for 15 is not 75 per cent. of 28, etc.

Complications.—The complications most frequently reported are: (1) Perforation peritonitis; (2) icterus; (3) duodenal stenosis and obstruction by cicatrization of the ulcer; (4) carcinomatous degeneration of the duodenal ulcer (ulcus carcinomatosum duodenale).

1. The frequency of *perforation peritonitis* has already been stated. The perforation may occur into the peritoneal cavity and into neighboring organs—for instance, the colon, pancreas, liver, or gall-bladder; it may break into arteries or veins, or into a closed and walled-off space within the abdomen. There are no specific symptoms peculiar to a perforating duodenal ulcer. Diagnosis is difficult. Bryant (*l. c.*), Brissaud (*l. c.*), Shield (*l. c.*), Lockwood (*l. c.*), and Lennander (*l. c.*) have undertaken operations for supposed perforation appendicitis which at the autopsy were proved to be perforated duodenal ulcer.

2. Icterus.—The presence of jaundice is of no differential diagnostic importance, because of the great scarcity of its occurrence as a complication of duodenal ulcer. Henoch has observed cases which were due to an actual duodenitis that had involved the papillæ of Vater. Collin (*l. c.*) could bring together only nine cases of this sort. As a rule, this complication runs its course under the clinical picture of catarrhal icterus.

3. Duodenal Stenosis and Obstruction.—This is also of very rare occurrence. As the ulcers are much more frequently located in portions of the duodenum nearest to the stomach, the resulting dilation resembles in all clinical and pathological pictures a dilation of the stomach; but if the cicatrization of the ulcer occurs lower in the duodenum, a dilation of this part of the bowel may be a consequence. Among 262 observations of duodenal ulcer, Collin (*l. c.*) reported gastric dilation 18 times and duodenal dilation 4 times. A cicatrix in the neighborhood of the ampulla of Vater may cause obstruction of the ductus choledochus and consequent icterus.

4. Carcinomatous Ulcer of the Duodenum.—Only four observations of this kind are reported in literature—those of Eichhorst (*l. c.*), Ewald (*l. c.*), Mackenzie (*l. c.*), and Schrötter (*l. c.*). All the complications thus mentioned offer almost insurmountable difficulties to efforts of diagnosis.

Treatment.—In all cases in which the physician has a suspicion of duodenal ulcer, as based upon the diagnostic signs mentioned in the preceding, he should associate himself at once with a competent surgeon and give him the opportunity to familiarize himself with the conditions of the case while diagnostic means of investigation are still possible. He may not need the operative interference of the surgeon, but similarly as in appendicitis the success of a possible operation will to a large extent depend upon the familiarity of the surgeon with the surrounding conditions and the condition of the patient himself. Whenever there are severe pains in the epigastric, right hypochondriac, and umbilical regions, associated with frequent occurrence of blood in the stools, the patient should be kept in bed and the stomach and upper bowels should be excluded from the digestive act entirely. This is done by feeding him by rectal enemata, as described in my work on "Diseases of the Stomach," second edition, pages 216 and 217. Hot cataplasms are placed over the painful areas, and the bowels are moved by small enemata of water. When absolute exclusion of the stomach from the digestive act has been kept up for about two weeks, a gradual return to a milk diet is cautiously undertaken. During the period of rectal feeding bismuth subgallate and subnitrate, five grains of each every three hours, and nitrate of silver, one grain three times a day, preferably in the form of a solution, should be given. If the latter is given in pill form, it is liable to pass through the duodenum undissolved; of course, if given in the form of a solution, the nitrate of silver may act first and most extensively upon the stomach. This can not well be prevented, but the escape of the nitrate of silver into the duodenum may be hastened by lying down on the right side and gently stroking the epigastric region from left to right. When there is evident hyperacidity, alkalis must be given.

The following formulas are the most available:

℞. Magnes. ustæ, ... gr. cl (10 gm.)
Sodii bicarb.,
Pulv. rad. rhei, ... aa gr. lxxv (5 gm.)
Ext. belladonnæ, ... gr. ivss (0.3 gm.).
M. Sig.—One-half of a teaspoonful three-quarters of an hour after meals.

Or:

R.	Sodii bicarb.,		
	Potass. carbonat.,		
	Magnes. ustæ,	āā gr. lxxv	(5 gm.)
	Ext. belladonnæ,	gr. iv	(0.25 gm.)
	Sacchar. lactis,	gr. ccc	(20 gm.).
M.	SIG.—One-half of a teaspoonful one hour after each meal.		

When pain is severe, opium or morphin should be used promptly. The majority of works on the subject refer the reader to some work on the treatment of gastric ulcer in the paragraph devoted to the treatment of the duodenal ulcer. There are, however, points of difference in the treatment which are of immense importance. In the first place, the duodenal ulcer peremptorily requires rectal feeding, exclusion of the stomach and duodenum from the digestive processes, and, whenever recognizable, should be treated from the onset as a surgical disease. Most authors agree that symptoms of duodenal obstruction, perforation, and subphrenic abscess indicate surgical interference. There can be no doubt about this, but there are extreme cases in which the operation is the *ultimato ratio*. In my opinion an operation should be done in all cases that present but two symptoms persistently—viz., great and continuous pain in the location repeatedly specified and severe intestinal hemorrhage. All such cases not getting well promptly after two weeks of the above treatment should be advised to subject themselves to exploratory laparotomy before any of the above severe complications have arisen. For the relief of symptoms strongly suggestive of intractable duodenal ulceration Codivilla ("Sperimentale," Mem. orig., vol. XLVII, pp. 4 and 6) resorted to gastro-enterostomy in two cases, with the result that both were cured. Lange ("Annals of Surgery," 1893) executed pyloroplastic operation successfully in a case of cicatricial stenosis caused by duodenal ulcer. For the rest of the surgical history of operative treatment of duodenal ulcer and its complications the reader is referred to the admirable article on "Perforating Duodenal Ulcers," compiled by the scholarly Robert S. Weir, in the "New York Medical Record" of May 5, 1900.

Intestinal Ulcers Occurring in Association with Cutaneous Burns.

The etiological relation and the course of acute duodenal ulcers occurring in association with cutaneous burns justify a short special consideration of this phenomenon. These ulcers occur mostly in the superior horizontal part of the duodenum. They may be present singly, but as many as from four to six have been observed together. They present an irregularly jagged form, and are generally much longer than they are broad. Nothnagel states the proportion of the length to the breadth as 5 to 1½ and 1½ to ½ mm. In this configuration the duodenal ulcer due to skin burns differs from the ordinary peptic duodenal ulcer. The duodenal complication develops very rapidly after the burn. It generally appears in from six to twelve days after the accident, but cases are reported in which the symptoms appeared in forty-eight hours, and death resulted in from thirty-six to forty-eight hours later. The latest onset of symptoms after the cutaneous burns is recorded as occurring in seventeen days. Greenwood ("Lancet," August 21, 1880) reported a case in which duodenal ulcer is alleged to have developed after the copious use of hot water, employed to arrest a secondary hemorrhage following amputation of the penis. The ulcers after extensive burns occur only exceptionally in the stomach, and very rarely in the lower sections of the intestine. The Fenwicks (*l. c.*, p. 87) state that superficial burns are followed by acute ulceration of the duodenum in about 6.2 per cent.

Historical.—McFarlane* was probably the first to point out the etiological relation of duodenal ulcer and cutaneous burns. Long, a well-known English surgeon ("London Med. Gaz.," N. S., vol. 1, p. 743), as far back as 1840 stated that clinically, as well as from their complicated effects on the internal organs, burns closely resembled acute febrile diseases of the skin, but Kerling, in 1842 ("Med. Chir. Transactions," 1842, vol. xxv), deserves the credit of having called attention to this etiological relation in the most emphatic manner.

Duodenal ulcers caused by *extended burns of the skin* have always commanded great practical and theoretical interest. According to the statement of Mayer, they usually develop in from seven to fourteen days after the burning; rarely earlier. Hunter, who bases his opinion on experimental observations, thinks that in the case of burns, the duodenal ulcers are caused by the fact that toxic substances formed in the skin, after having been taken up in the blood, are secreted with the bile into the duodenum, and produce a form of necrobiosis of its mucous membrane. Other authors since that time have assumed a thrombosis of the duodenal veins as the cause of duodenal ulcers when the skin has been burned. This thrombosis is thought to be caused by the liberation of the fibrinogen. Whereas the other duodenal ulcers occur much more frequently in men than in women, the opposite is true in the case of the ulcers caused by burns on the skin.

---

*Cited by Nothnagel. He does not give the reference.

Pathological Physiology.—The various theories that have been propounded in explanation of the visceral changes occurring in extensive burns have been compiled in an interesting paper by Charles R. Bardine, "Journal of Experimental Medicine," volume II, page 501. The changes of the blood that have been recognized and confirmed are the following: (1) There is a marked leukocytosis, depending chiefly upon an increase of the polymorphonuclear cells; (2) the specific gravity of the blood is slightly increased; (3) the erythrocytes are injured physiologically and morphologically. A fair consensus of opinion also exists concerning the effects of the altered blood after skin burns on the tissues of the body. They may be summarized as follows: (1) In the cells of the spleen, bone-marrow, and lymphatic glands fragments of red blood-corpuscles are collected, and blood pigments have been observed in the epithelium of the kidneys; (2) thrombosis has been observed in the capillaries and veins of some of the viscera. The question what causes these ulcers after skin burns to develop most frequently in the duodenum, and very rarely in any other part of the digestive canal, is difficult to answer, and thus far we have only hypotheses in explanation of this strange relation between the duodenum and the skin. W. Hunter found duodenal ulcers occurring after subcutaneous injections of toluylendiamin ("Pathological Transactions," 1890). He suggested that toxic substances were excreted into the duodenum with the bile after skin burns similarly as after subcutaneous injection of toluylendiamin. The other hypotheses are given in the paper by Bardine (*l. c.*), but none of them explains why the duodenum should be the prevalent location of the ulcers while the stomach is comparatively immune. According to Ponfick, Wertheim, and Max Schulze, these ulcers are caused by hemorrhagic infiltration and destruction of red blood-corpuscles. The Fenwicks (*l. c.*, p. 87) state that frost-bite has also been cited as a cause of this type of ulcer. Förtser ("Handb. d. path. Anat.," Leipzig, 1864) has made an observation of this kind, and J. Krauss ("Das perfor. Geschwür i. Duodenum," Berlin, 1865) has ventilated the question whether duodenal ulcers may not occur after injuries due to freezing.

The theories that explain duodenal ulcerations after burns assume that altered conditions in the affected areas give rise either to poisonous substances or to substances which, after being absorbed, cause the internal organs to produce toxic material. Kijanitzin ("Virchow's Archiv," vol. CXXXI, 1893, p. 436), by treating the blood of variously burnt dogs by a modification of Brieger's method, obtained substances which he claimed very much resembled the peptotoxins of Brieger. Bardine made a study of the organs from the bodies of five children fatally burnt. The chief lesion seen in the intestines and stomach was swelling of the lymphatic tissue, which contained focal necroses and nuclear fragments. Duodenal ulcers as such were not observed. The main changes in the intestine were the parenchymatous degenerations in the lymphatic glands. The lesions described by Bardine are compared to, and described as being essentially like, those produced experimentally in animals by the injection of diphtheria toxin (Welch and Flexner, "Bulletin of the Johns Hopkins Hospital," 1891); indeed, the changes seen in all the internal organs were very similar to those observed after fatal cases of fevers that exert their chief deleterious effects by toxic substances circulating in the blood.

The focal lesions in the intestinal lymphatic glands described by Bardine may well lead to a form of ulceration sometimes described under the name of follicular, which, when once started, may be further enlarged by the process of autodigestion, being superadded to a toxic inflammation in the glands of Lieberkühn and Brunner. This would also to some extent explain why such ulcers should occur most frequently in the duodenum, because, in the first place, the glands of Brunner are most frequent in this region, and, secondly, because the process of autodigestion can occur here with an intensity which is possible nowhere else in the intestinal canal.

Symptoms.—The symptoms of duodenal ulcers of this type are sometimes entirely masked by the gravity of the symptoms of the burns themselves. As duodenal ulcers occur only in 6.2 per cent. of cutaneous burns, the diagnosis of the duodenal complication will not be certain before hemorrhage from the stomach or the bowel has occurred, or before the appearance of the symptoms of perforation peritonitis.

Prognosis.—The prognosis is always grave. I could find no report of cases in literature of well-authenticated duodenal ulcer following cutaneous burns which recovered. In a number of so-called recoveries the diagnosis was doubtful. Three cases of this type (severe pain in the left hypochondrium in association with intestinal hemorrhages after cutaneous burns) which I saw in consultation with other physicians all terminated fatally. The possibility of operation was thought of, but in view of the shock resulting from the burn it was considered doubtful whether the patient would survive operative interference.

Treatment.—The treatment will be that given under peptic duodenal ulcer, but it will be more especially a treatment for intestinal hemorrhage and perforation peritonitis.

Embolic and Thrombotic Intestinal Ulcers.

The small amount of blood in the intestinal vessels is a striking feature observable at most autopsies, especially if the cardiac action was considerably weakened for some time prior to death. This disposition of the intestine to anemia is apparent even under conditions in which we would expect a hyperemia, for in many cases in which an intense exudative inflammation with all its clinical symptoms was present during life, one finds in the postmortem examination very little and often no hyperemia. In some cases that have presented such symptoms one is surprised to find an extreme paleness of the intestine. Personally I have a record of cases in which I saw the intestinal mucosa at laparotomies done for the arrest of profuse intestinal hemorrhage, where the mucosa was swollen, very much congested, and in places even presented indications of infarcts. At the section which occurred in one case twenty-four hours after operation, this hyperemia had entirely subsided and given place to a condition that could justly be termed anemic. Orth ("Specielle patholog. Anatomie," Bd. 1, S. 772) states that he was struck with this anemia at autopsies after the summer diarrheas of infants.

Notwithstanding this prevailing condition, visible inflammatory hyperemias occur in the intestines after death, and are generally localized on the valvulæ, the villi, or around the follicles. All intestinal ulcers are, as a rule, surrounded by a hyperemic area. A very marked and extensive anemia is one of the characteristic signs of advanced amyloid degeneration of the intestines.

Embolism of the main stem or the larger branches of the mesenteric artery produces a clinical and pathological picture so widely differing from the effects of occlusion of the smaller branches of this artery, which run along in the intestinal wall itself, that a separate consideration of embolism of the larger branches becomes necessary in a special chapter. Occlusion of the minuter arterial branches in the intestinal walls may lead to changes eventuating in circumscribed necroses, which may aptly be termed embolic ulcers. The origin of the embolus can generally be traced to one of three underlying conditions: (1) Arterial sclerosis, especially atheromatous degeneration of the aorta; (2) endocarditis; (3) pyemia with probable pulmonary abscesses. The pathological features vary according to the size of the vessel that is stopped up and according to the condition of the embolus, which may be a simple or sterile clot or may be septic. When one of the very smallest vessels is occluded by a simple sterile embolus, it may be of no consequence whatever and give no clinical or pathological indications whatever. Occlusion of a mesenteric branch of the intestinal vessels as a rule causes no functional disturbance. Occasionally small wedges of infarction, the broad base of which is directed toward the mucosa, the apex lying in the submucosa, have been observed in the intestine. Orth (*l. c.*, p. 775) states that he has examined completely organized emboli in the intestinal arteries where no trace of any change of infarct could be discovered. An infarct of the mucosa may, but rarely does, develop in connection with obstruction of the very minutest vessels. It generally leads to a very slight swelling or a hyperemic redness of the surrounding tissue; but emboli in larger vessels cause a small round-cell infiltration of the area supplied by the branch; hemorrhagic infiltration follows, and in the central portion of this necrosis eventuates. Embolic ulcers then develop by necrotic degeneration within the region formerly supplied by the occluded vessel. The ulcers are, as a rule, small, may have a round configuration, or encircle the lumen of the bowel partially in the form of a girdle, or they may have an entirely irregular outline. This process, as observed in the cecum by Parenski, involved five folds which were hyperemic, swollen, firm and rigid, of an almost black color, and infiltrated with blood.

The submucous tissue corresponding to these areas was similarly infiltrated, and in between the folds there was a purulent necrobiosis of the mucosa. The corresponding portion of the peritoneum was swollen and presented a bloody suggillation. When the necrobiotic process is rapid, which is usually the case, the entire thickness of the intestinal wall may become involved, and a fibrinopurulent, perforative peritonitis is the result. In the neighborhood of these necrotic portions croupous or diphtheric inflammations of the mucosa are occasionally found in the intestine itself. When one considers the nature of the processes which bring about embolic or thrombotic ulcers, it is natural that we do not, as a rule, find them occurring singly, but several of them together; and that, in addition, infarctions in other organs can be detected—the spleen or kidneys, for instance. Septic processes may result in capillary emboli of a pronounced character. Small miliary abscesses consisting of accumulations of densely crowded round cells surrounding a blood-vessel occupy the submucosa. This accumulation of round or pus-cells generally extends from the peritoneum to the mucosa, which is in some cases destroyed, and thus an embolic ulcer is formed. Nothnagel (*l. c.*, p. 156) narrates a case of capillary emboli of the intestinal walls of a septic character originating from a pulmonary abscess. The patient, a female, suffered from cutaneous infiltrations with formation of small abscesses in various parts of the body as a result of years of continued hypodermic injections of morphin. The axillary glands had suppurated. The patient had albuminuria, and died with symptoms of pyemia. At the autopsy small pus foci were found in the large white kidneys, abscesses in the lungs, and numerous miliary abscesses in the intestines. A portion of the latter were changed into ulcers. Parenski reports a case in which thickly indurated cicatrices of healed ulcers, which were darkly pigmented and permeated the entire intestinal wall, were found in the lower portion of the jejunum. The accompanying clinical and pathological conditions made it probable that they were of embolic origin.

Thrombotic Intestinal Ulcers Occurring in Multiple Degenerative Neuritis (Infectious).—It is logical to classify these intestinal lesions with the ulcers originating from circulatory disturbances, because changes have been demonstrated to occur in the smallest arteries, eventually leading to occlusion of the vessels during the course of multiplex infectious and degenerative neuritis. Such a condition of things has been described by Lorenz in multiple neuritis. There were numerous losses of substance in the jejunum, extending over large areas. Part of the ulcers involved the peritoneum. In addition there were very minute losses of substance, the edges of which were formed by swollen and injected mucosa and the base by an irregular, dark-brown, softening tissue. The arterial mesenteric branches corresponding to these areas were thickened and the lumen almost obstructed.

Minkowski has given a graphic account of this form of intestinal ulcer. The intestinal mucosa of his case of multiple neuritis exhibited a slate-colored pigmentation and was permeated with very numerous ulcerations which occupied the entire circumference of the intestinal lumen and did not appear restricted to the follicular apparatus. The ulcers presented an irregularly zigzag margin, some of them located with their long axis in the direction of the long axis of the intestine, and others situated obliquely. One of the losses of substance had led to a perforation, while several of them had penetrated to the muscular coat. Minkowski attributed the ulceration to the vascular disease and the consequent interference of circulation, for he was able to demonstrate thrombi and obliterated vessels in the environment of the ulcers. Acland and Hale White have suggested the occurrence of so-called "trophic" intestinal ulcers in the course of spinal diseases, which ulcerations they compare to the development of bed-sores. They do not, however, present satisfactory clinical evidence that such an intestinal complication of the spinal lesions is at all probable. Finally, a case reported by Kussmaul and Maier (*l. c.*) might be classed with this group. These authors originally designated the clinical and pathological picture described by them as due to "peri-arteritis nodosa." The mucosa of the small and the large intestine presented numerous areas of necrobiosis and superficial losses of substance, inflammatory infiltration, and ecchymoses associated with stagnation. They emphasized the similarity of the clinical and pathological features to the effects of embolism of the mesenteric arteries. My personal experience with embolic intestinal ulcers is limited to a female patient who, during life, presented irregular attacks of diarrhea containing pus and necrotic tissue in the evacuations, so that the diagnosis of pseudomembranous dysentery had been made. At the autopsy I found eight ulcers in the cecum, varying from the size of a pea to a large irregular ulcer about four centimeters long and about one centimeter broad. There was a large gonorrheal abscess involving the left tube and ovary; the appendix was normal. Minute emboli which contained pus cocci were found in the minuter arteries of the submucosa. None of the ulcers had produced a perforation, but several of them had their bases in the peritoneum. The remaining abdominal and thoracic organs were normal.

Amyloid Ulcerations of the Intestine.

Amyloid degeneration of the intestines as a whole will be described in a separate chapter, to which I refer for the more generally accepted facts concerning the nature of this process.

Amyloid intestinal ulcers are of very rare occurrence. I base this conclusion not only upon a large number of personal observations at autopsies in various hospitals of Baltimore, but also upon the scarcity of well-authenticated cases in literature. Courtois-Suffit ("Traité de Méd.," by Charcot, Bouchard, and Brissaud, tome IV, p. 487) remarks that these ulcerations are not rare, and Colberg ("Deutsch. Arch. f. klin. Med.," Bd. II, 1867, p. 478) even declares them to be frequent. He explains the mode in which such ulcers begin by assuming that the villi become rigid and brittle in consequence of the amyloid condition of their capillaries, and thus prone to break down from the mechanical violence of the passage of the intestinal contents. As amyloid degeneration of the kidneys generally coexists, the intestinal mucosa is called upon to excrete the products of urea decomposition, and thus the chemical action of ammoniacal compounds is superadded. Most observers agree that the arteries and capillaries of the mucosa and submucosa, particularly of the villi, are degenerated; but the entire tissue of the villi may completely undergo amyloid degeneration. As the amyloid substance is very brittle after death, the villi are frequently observed to be defective. This, however, does not permit of the conclusion that such fragility of these structures already exists during life, and that they could be broken off by the mechanical violence of the passage of the intestinal contents, as Colberg assumes. Ulcers of this type appear to occur in all regions of the intestinal canal, varying in size from that of a lentil to extensive losses of substance extending circularly around the lumen of the intestine and extending from five to fifteen centimeters in length. Eberth ("Virchow's Archiv," Bd. LXXX, S. 166) and Kyber (*ibid.*, Bd. LXXXI, S. 305) have studied the condition of the intestinal epithelium in this process. Eberth found no epithelial cells and Kyber exceedingly few of them in a condition of amyloid degeneration. The edges of the ulcers are usually smooth and slightly thickened, while the base is pale and in some instances shows striations which have been attributed to the rigid walls of blood-vessels underneath the ulcer. Portions of the intestine between the ulcers show amyloid degeneration but no inflammatory changes. Colberg asserts that nowhere in such ulcerations is there any tendency to healing or cicatrization. I have emphasized elsewhere the extreme pallor and anemia which are characteristic of amyloid intestine. This form of degeneration may be recognized also by the waxy shine, the thickening, and in places by the broken appearance of the villi. The only test of any reliability, however, is the iodin reaction, which demonstrates the degeneration in the villi and the mucosa, while the Peyer's patches and the follicles, as a rule, give no amyloid reaction or only a very slight one. The amyloid ulcers themselves do not give the amyloid reaction with iodin. It is very probable that with more exact application of the staining for tubercle bacilli in so-called amyloid ulcers it will be found that the majority of losses of substance usually attributed to this form of degeneration are of a tuberculous nature.

Amyloid ulcers possess little or no clinical significance. What there is of interest concerning the signs and symptoms will be considered in the general chapter on Symptomatology of Intestinal Ulcers.

ULCERATIONS DUE TO INFLAMMATORY PROCESSES.

Catarrhal Intestinal Ulcers.

The catarrhal inflammations described in the chapter on Enteritis may lead to the formation of intestinal ulcers, which have been variously designated as catarrhal or follicular ulcers, according to their origin, which is generally from the tissue of the mucous membrane or from a follicle. Both of these types occur as well in acute as in chronic enteritis. The separation into catarrhal and follicular ulcers is justifiable for anatomical reasons only. Both may occur simultaneously during the same catarrhal process, and clinically no separation is possible. They are most frequently found in the colon, and are rare in the small intestine.

Catarrhal Ulcers not Involving the Solitary Follicles.—These owe their origin to a superficial loss of epithelial cells, which may be brought about by toxic or irritating chemical substances penetrating into the mucosa from the lumen of the intestine, or passing from the intestinal vessels or tissue into the lumen,—the passage either way will eventually injure the epithelium,—or by direct injury exerted by bacteria, intestinal parasites, etc. An insignificant loss of epithelial cells makes possible the invasion of bacteria and penetration of chemically irritating substances. The superficial layers of the mucosa undergo necrobiosis; an accumulation of lymphoid elements by which the superficial portions of the glands of Lieberkühn are entirely obscured precedes the development of a superficial erosion. Woodward (*l. c.*, p. 573) has presented us with a very exact account of the pathogenesis and histology of these types of ulcers, profusely illustrated by excellent drawings and photographs. According to him the simple catarrhal ulcers always originate from the surface of the mucosa. In this they differ essentially from embolic intestinal ulcers which originate in the submucosa.

While Rokitansky and Leube state that the inflammation of this type may as well originate on the surface as in the interior of the mucosa, Nothnagel agrees with the views expressed originally by Woodward. The incipiency of the catarrhal erosions in the manner just described is soon followed by a separation and detachment of the columnar epithelium of the mucosa, leaving an abraded surface composed almost entirely of round cells and lymphoid elements, presenting the appearance of the surface of a granulating wound. The reticulum of the adenoid tissue breaks down and softens, and the round cells are carried off as pus-corpuscles. Ulcers of this type may be so superficial that only the upper half of the glands of Lieberkühn are destroyed, while the bases of these glands can still be recognized beneath the ulcerated surface. But in other instances the round-cell infiltration pervades the submucous connective tissue and may even penetrate to the serosa. Several small ulcers may, by coalescence, develop a large one. Perforation may occur, but seems to be rare from this type of ulcer. The edges of the catarrhal ulcer are tumefied by hyperemia, infiltration with pus-cells, and the columnar surface epithelium near the edges is frequently undermined. This undermining process I take to be of a mechanical nature, since the submucosa is more readily destroyed by the inflammatory necrobiosis than the epithelium, the ulcer spreading more readily under the columnar cells of the surface, which must necessarily overlap the undermined portion. Between the various ulcers the mucosa presents the swollen catarrhal condition described in the chapter on Enteritis.

Follicular Ulceration (Enteritis Follicularis).

When the cells of the follicles have become diseased under the influence of the catarrhal inflammation, the invasion of the bacteria is facilitated by the lessened power of resistance. Whether or not follicular inflammation can arise in the absence of bacterial invasion is a question which I am not prepared to answer, and the investigation of which presents many technical difficulties. But no matter what the original cause of the lessened power of resistance of the follicular cells, the detrimental influence of the bacteria is eventually superadded. An accumulation of round and lymphoid cells in the parenchyma of the follicles follows the infection. In the small intestine the enlarged follicle frequently protrudes beyond the mucosa like a tiny polypus before ulceration occurs; but in the colon the follicles, as a rule, ulcerate before any considerable enlargement occurs. Occasionally, however, the solitary follicles of the colon, also, may attain the size of a bird-shot before they ulcerate. Woodward has described two ways in which the ulcerative process in the enlarged colon follicles begins. In the first case, the central portion of the little tumor softens, the reticulum of its parenchyma becomes granular, loses its cohesion, liquefies, the lymphoid elements are set free as pus-corpuscles, and a small abscess results, which ruptures at its apex, the drop of pus escaping upon the mucous surface, leaving a tiny cavity, which is a follicular ulcer. In the second, ulceration begins at the apex of the enlarged follicle by the formation and separation of a minute slough, or by the liquefaction of the reticulum and the floating away of the lymphoid elements as pus-corpuscles; in this way it invades the follicle from the surface, with the same ultimate result as before. The necrobiotic changes ultimately destroy the whole follicle. Both the superficial catarrhal ulcer as well as the follicular ulcer may heal by cicatrization, which may result in stenosis of varying degree. It is not always possible to distinguish between follicular and nonfollicular ulcers. Small follicular ulcers may spread into large erosions. Woodward describes a third form of origin of catarrhal ulcers in chronic enteritis in addition to the follicular and nonfollicular ulcers. This form, according to this author, is due to a peculiar cystlike development of the glands of Lieberkühn, a cystic transformation of hypertrophic gland tubules in the parenchyma of the solitary follicles. When such cysts rupture and discharge their contents, the cavities left closely resemble ordinary follicular ulcers.

Stercoral or Decubital Ulcers.

In the chapter on Constipation the stercoral ulcerations, which owe their origin to the pressure of hardened, stagnating feces, have already been described. They are essentially catarrhal ulcers caused by necrosis of the superficial layers of the mucosa and subsequent purulent inflammation of the submucosa. These ulcers develop most frequently in those localities which are most exposed to the pressure of the hardened fecal masses: for instance, in the flexures of the colon,—the hepatic and splenic,—in the cecum and appendix, and in the sigmoid flexure and the rectum. Concerning the influence of the rectal valves in retarding the descent of the fecal column the reader is referred to a practical monograph on "Disorders and Diseases of the Rectal Valves," by Thomas Charles Martin ("Obstipation," Phil. Med. Pub. Co., 1899). Concerning the influence of stercoral ulcers on the production of appendicitis see Illoway, "Constipation," New York, 1897, page 136. Stercoral ulcers are also common in association with stenoses of the intestine, where they occur only above the obstructed portion, while the mucosa below the stenosis is normal. This type, like other intestinal ulcers, may cause intestinal strictures by the cicatricial contraction during healing. Grawitz, who originally suggested the term "decubital ulcers" for this type, remarks that such cicatricial intestinal strictures have been erroneously attributed to carcinoma or syphilis. Intestinal stenosis due to constrictions from healing stercoral ulcers are very rare, but there can be no doubt that such cicatrices in the intestine may become carcinomatous (Nothnagel), similarly as carcinoma development has been demonstrated in the cicatricial tissue of healed gastric ulcers.

ULCERATIONS DUE TO ACUTE INFECTIOUS DISEASES.

The acute infectious diseases in which intestinal ulcerations have been observed are typhoid or enteric fever, yellow fever, dysentery, diphtheria, anthrax, septicemia, erysipelas, and variola. Information concerning these will probably not be sought in a special work on diseases of the intestines. The intestinal ulcerations of dysentery and those occurring in diphtheria have already been described in the chapter on Dysentery and Diphtheric Enteritis. In typhoid fever, dysentery, and sometimes in diphtheria the intestinal ulcerations belong to the characteristic features of the infectious process; but in anthrax, sepsis, erysipelas, and varioloid the intestinal ulcerations are the great exception in the course of these diseases.

Anthrax.

The gastro-intestinal lesions of anthrax are rare and have little clinical significance in our country. The clinical features are described by F. von Koranyi (*Zoonosen*, Nothnagel's "Spec. Pathol. und Ther.," Bd. v, Th. 5, S. 36).

Septicemia.

Intestinal ulcers in septicemia are of extremely rare occurrence. In 27 cases of septicemia that came to autopsy from Nothnagel's clinic not a single case of ulceration in the intestine was found.

Billroth has reported an interesting case of a male patient who was afflicted with a fatty heart and hepatic cirrhosis upon whom an operation for goiter was performed. Two days thereafter intestinal symptoms and bloody evacuation followed, and on the sixth day, under constant rise of temperature (to 40.1° C.), death occurred. At the autopsy several ulcers were found in the duodenum. As the intestinal mucosa is found hyperemic, inflamed, and in places exhibiting hemorrhagic infarcts during the course of experimental septicemia, Billroth attributed the duodenal ulcers to this condition. Admitting, however, that in this patient additional factors—the fatty heart and the hepatic cirrhosis—may have caused passive congestion in the intestine leading to autodigestion and ulcer formation, one must agree with Nothnagel that even according to this explanation the duodenal ulcers in Billroth's case (*l. c.*) can not be directly attributed to the sepsis. This could be claimed only when such complications as were mentioned in this case are absent. Intestinal ulcers occurring in septicemia can logically be attributed to a hemorrhagic septic intestinal catarrh eventuating in local necrobiosis of the intestinal mucosa (Nothnagel). The same can be said of the pathogenesis of intestinal ulcers which have been reported to occur especially in the duodenum in association with erysipelas. Larcher and Petitbien (*l. c.*) have observed this intestinal complication of erysipelas.

Variola.

In variola catarrhal enteritis and hemorrhages are frequent; secondary inflammatory swelling of the mesenteric glands has also been reported. Ulceration of the follicles occurs, but is a rare complication. It is not known whether these intestinal ulcerations in variola originate from typical pustules.

Intestinal Ulcers in Association with Skin Eruptions.

In a number of skin diseases efflorescences have been observed in the mucosa of the mouth which were histologically identical with the characteristic skin eruptions (see Hans Herz, "Die Störungen des Verdauungs-Apparates," u.s.w., S. 496), and it is presumed that the exhaustive diarrheas and other intestinal symptoms may depend on the same disease involving the intestinal mucosa, but nothing definite pathologically has been published on this subject. Nothnagel states that intestinal ulcers have been observed in acute pemphigus, and, according to Lapponi (*l. c.*), similar ulcers have occurred in association with pellagra. The etiological relation and the pathogenesis in these cases are obscure.

ULCERATIONS OCCURRING IN CHRONIC INFECTIOUS DISEASES.

Tuberculosis.

Tuberculous Intestinal Ulcers.—The earliest reliable historical account of tuberculous intestinal ulcer is by G. L. Bayle ("Rech. sur la Phthisie Pulmonaire," Paris, 1810, p. 59). In 100 postmortem examinations on subjects dead from phthisis he reported to have found 67 cases of ulceration in the intestine. Bayle's earlier contributions to the subject of tuberculosis are contained in Corvasart's "Journal de Médecine," from 1803 to 1805. The further history of the literature is given in foot-notes by Woodward (*l. c.*, p. 576).

Tuberculous intestinal ulcerations have been very carefully studied, and their etiology and histology are now quite thoroughly understood. Excepting typhoid fever and dysentery, tuberculosis is the most frequent cause of ulceration in the intestines. The distinction between primary and secondary tuberculous intestinal ulcerations is a logical one. The primary form occurs as an original infection in the intestine in individuals otherwise free from tuberculosis; the secondary form is that occurring in association with other tuberculous affections elsewhere in the body. The primary intestinal form is rare in adults and occurs more frequently in early childhood, being then an infection due to feeding with tuberculous milk or meat. Klebs (*l. c.*) and Leube (*l. c.*) have denied the existence of primary intestinal tuberculosis, but Bollinger (*l. c.*), Eisenhart (*l. c.*), Melchior (*l. c.*), and Wyss (*l. c.*) have established the occurrence of this form on the basis of irreproachable investigation. Secondary intestinal tuberculosis occurs most frequently in association with pulmonary phthisis. Concerning the frequency of intestinal tuberculosis, the statements of various observers differ considerably. Herxheimer (*l. c.*) found 57 cases of intestinal tuberculosis in 58 cases of phthisis—that is, only one of these cases of phthisis failed to show the intestinal complication. Weigert (cited by Herxheimer) states that 90 per cent. of cases of phthisis have intestinal tuberculosis. Höning gives 70 per cent., and Frerichs (*l. c.*), in 250 cases of chronic phthisis, found tuberculosis of the ileum in 200. The colon was implicated in 115 cases; the rectum in 18; the colon alone was tuberculous in 8 cases. Expressed in percentages, Frerichs' observations were as follows: Ileum, 80 per cent.; colon and ileum, 45 per cent.; colon alone, 3 per cent.; rectum (always secondary), 7 per cent. Steiner and Neureutter (*l. c.*) found intestinal tuberculosis 71 times in 302 autopsies on children. Among these there were 176 cases of pulmonary tuberculosis and 275 cases of tuberculosis of the bronchial glands. Wiederhofer (*l. c.*) found 101 cases of intestinal involvement among 418 cases of children dead of tuberculosis. The small intestine was tuberculous in 98 cases; the large intestine alone in 3 cases; the large and the small intestine in 22 cases. The principal tuberculous diseases of other organs in association with the bowel lesions as stated by him were as follows: Pulmonary phthisis, 76; tuberculosis of the brain and membranes, 14; granulation tuberculosis of the lungs, 8; tuberculosis of the glands, 3 cases; tuberculosis of the mesenteric glands, 83 cases. Among the last there were 48 cases of tuberculosis of the intestines coexisting with that of the mesenteric glands. The following is the table given by this author concerning the age of these 418 children:

AGE.	0–1.	1–2.	2–3.	3–4.	4–5.	5–6.	6–7.	7–8.	8–9.	9–10.	Over 10.
General tuberculosis.	23	54	69	62	47	31	26	26	14	15	51
Intestinal tuberculosis.	3	3	14	14	15	4	9	8	2	5	24
Tuberculosis of the mesenteric glands.	4	4	17	12	13	5	9	7	1	4	7

I have made an investigation of the records of autopsies at Bay View Hospital, the University of Maryland Hospital, the Maryland General Hospital, added a number of necropsies occurring in practice, and compiled the records of 312 autopsies on sub-

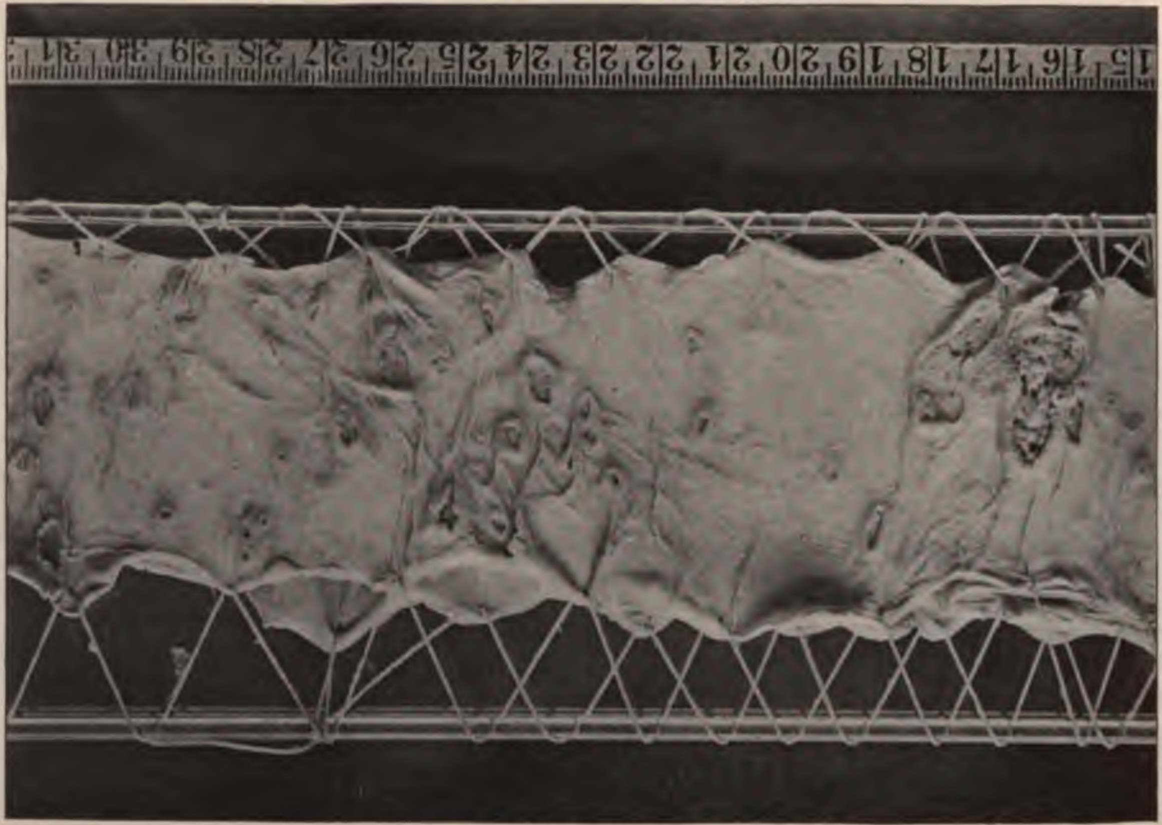


FIG. 31.—TUBERCULOUS ULCERATION OF ILEUM.

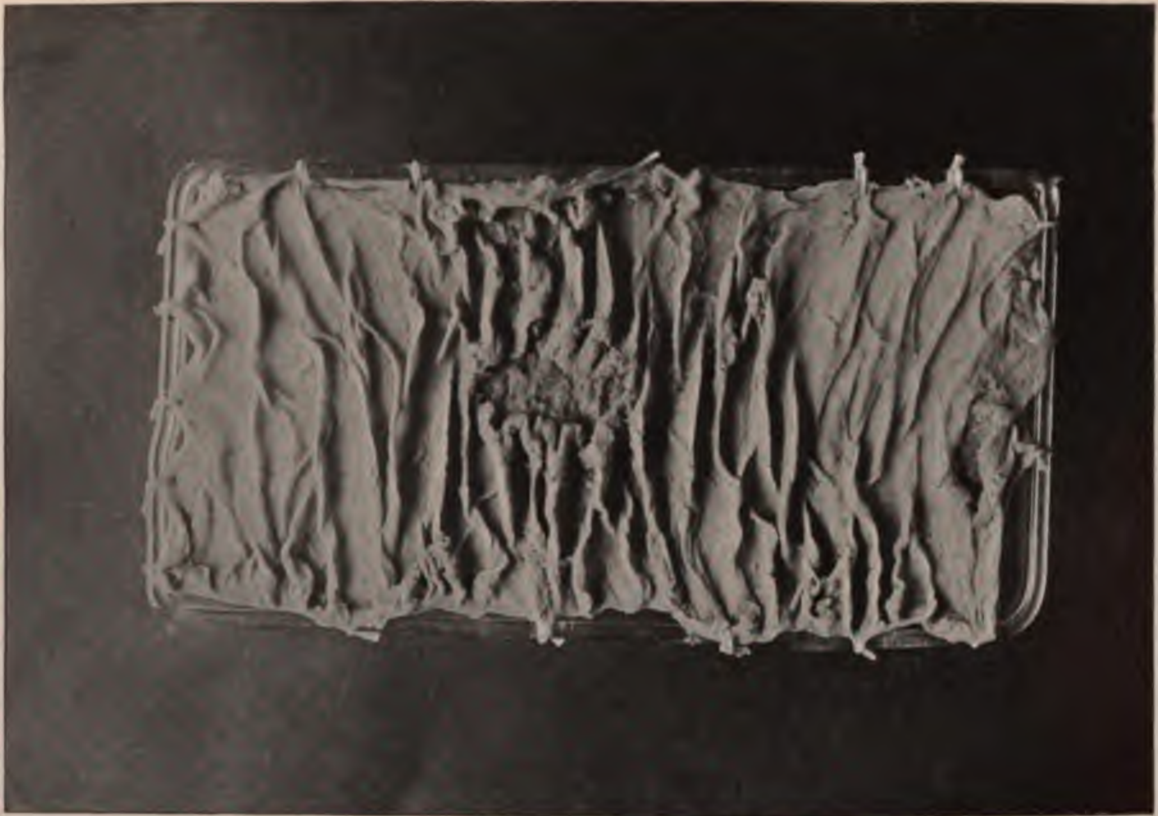


FIG. 32.—TUBERCULOUS ULCERATION OF ILEUM.

jects dead of tuberculosis. I found 97 cases in which reference was made to the condition of the stomach, intestines, and mesenteric glands. Of these, 80 cases had tuberculosis limited to the ileum and jejunum; 6 cases are reported to have had tuberculosis limited to the colon; in 10 cases the colon and ileum alone were involved; in 1 case, at which I personally attended the autopsy, the duodenum alone was involved. There were 60 cases of tubercular mesenteric glands, in 3 of which there were no intestinal lesions discoverable.

The most frequent seat of tuberculous ulcers is the ileum, particularly the lower portion, just before its entrance into the cecum. They occur also in the jejunum and duodenum, but are not so frequent here as they are in the lower portion of the bowel. The predilection for these sections of the intestine may be partially explained by the anatomical fact that the lymph-nodules, which play an important rôle in the colonization of the tubercle bacillus, are most numerous in the lower portion of the ileum, and also that the intestinal contents remain longer in this place than in localities higher up in the intestine. The chemical composition of the intestinal contents also plays a rôle in favoring this predilection, for here the reaction of these contents begins to be less acid, and consequently less antiseptic, than in the upper portion of the intestines. It also contains a larger percentage of toxic products which in themselves are injurious to the epithelium of the mucosa.

In the chapter on "Tuberculosis" in my work on "Diseases of the Stomach" I have referred to the comparative immunity which the stomach enjoys from tuberculosis. A similar condition, while not to so marked an extent, exists in the intestine, for in secondary intestinal tuberculosis the changes in the intestine are, as a rule, not nearly so advanced nor so pronounced as the secondary changes in other organs. In experiments made on guinea-pigs by feeding them with tuberculous masses, Orth (*l. c.*) found only 7 instances of intestinal tuberculosis in 9 animals that had become generally tuberculous, but even in these 7 cases the intestinal changes were not nearly so pronounced as the general tuberculosis in the bodies of the animals. The investigations of Dobroklonsky (*l. c.*), Tchitschowisch (*l. c.*), and Fischer (*l. c.*) also show that a transportation of tuberculosis to the intestinal mucosa may occur with an entirely intact epithelium.

Gottsacker (*l. c.*) asserted that miliary tubercles do not develop at the beginning of the infection, but follow a chronic inflammation and caseation of the follicles and granular proliferation of the perifollicular tissues. Höning (*l. c.*) is of the same opinion, but goes a step further by saying that he never found tubercle bacilli before the follicles had disintegrated. The incipient swelling of the follicle is not of a tubercular nature; according to these writers, necrobiosis of these intestinal structures is necessary before the bacilli can find a nidus. The far more important and authentic investigations of Orth (*l. c.*) and Baumgarten (*l. c.*) show conclusively that the caseation of the intestinal follicles is of a tuberculous nature from the very beginning, for Baumgarten found giant cells and tubercle bacilli in the unopened follicles, and Orth states that tubercle bacilli can be found in the unopened intestinal follicles as well as in the tumefied edges and in the bases of the ulcerations. They are most numerous in the actively progressing ulcers.

Etiology and Pathogenesis.—Intestinal tuberculosis is, as a rule, a secondary affection occurring after other organs, particularly the lungs, have developed tuberculosis. The efforts to show that the intestines are comparatively immune from the disease, even after demonstrable dissemination of tuberculous infection through the blood, are not as yet supported by satisfactory experimental and pathologic evidences. It is established experimentally that the tubercle bacillus may penetrate into the mesenteric glands and there develop further without causing any alteration in the intestinal wall. Following this, tubercles may appear in the liver while the intestines and lungs are intact (Cornet, "Die Tuberculose," in Nothnagel's "Spec. Pathol. u. Ther.," Bd. XIV, 2. Hälfte, S. 60). It is evident that it must be impossible to determine the relative frequency with which the intestines constitute the portal of infection of the tubercle bacillus from any lesions in the bowel-wall itself. It is necessary to include all possible lesions of the mesenteric and omental glands in counting up the tubercular lesions of the intestines. It is probable, therefore, that the intestines are much more often the portals of entry of the infection than has been hitherto su
From the standpoint of prophylaxis, it is very important to estab-

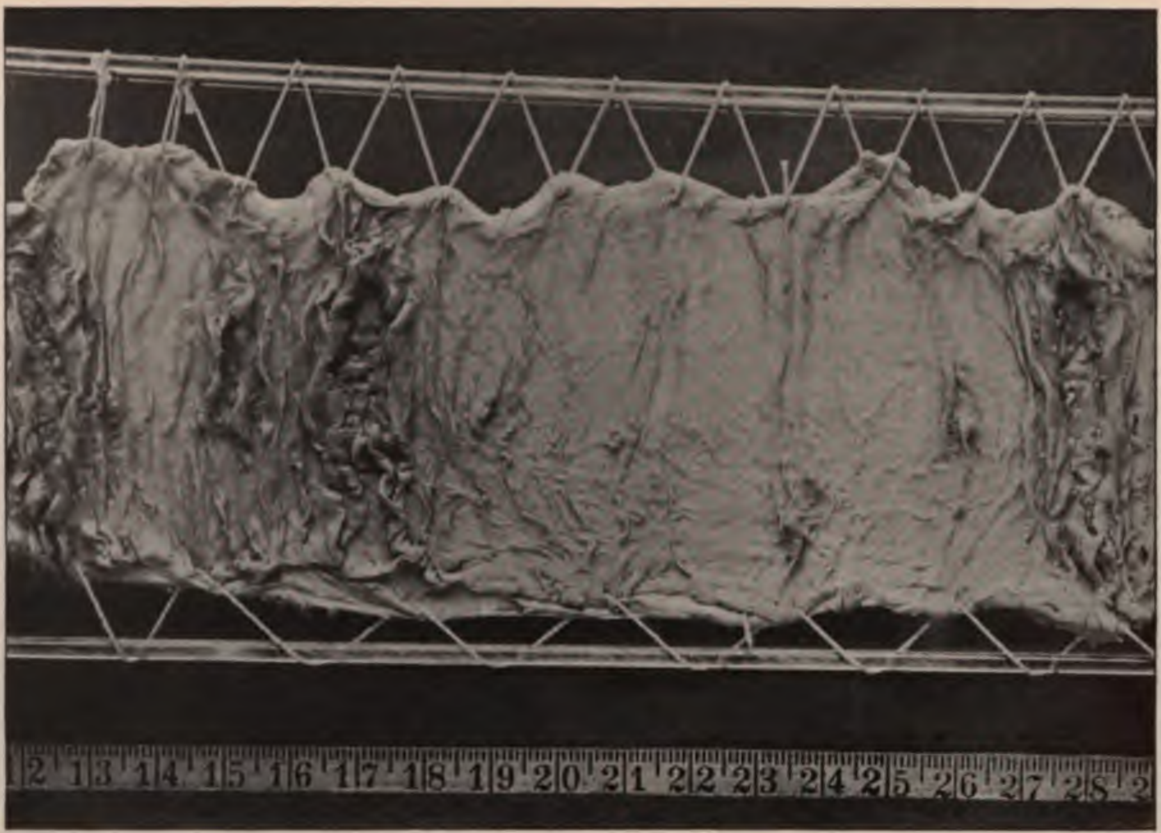


FIG. 33.—TUBERCULOUS ULCERATION OF ILEUM.

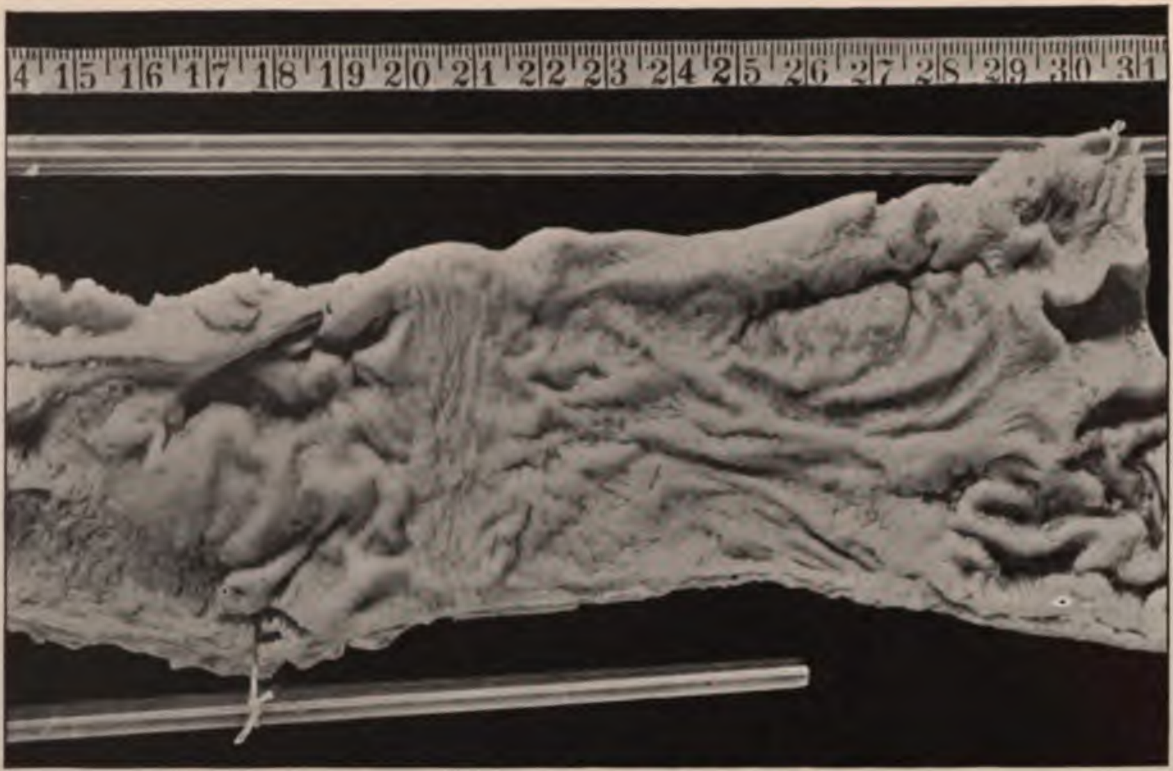


FIG. 34.—TUBERCULOUS ULCERATION OF SIGMOID FLEXURE.

lish the agency of the intestine as a portal of entry.* Klebs was the originator of the explanation that the swallowed tuberculous sputum was the source of the infection of the gastro-intestinal tract. It is undoubted that tubercle bacilli may be imported into the intestine along other routes—for instance, tuberculous lymph-glands may break into the esophagus or intestine itself. The primary tuberculosis occurs most frequently in children, and is generally a food infection (Fütterungs-Tuberkulose). The causation of intestinal tuberculosis by swallowed sputum should emphasize the necessity of guarding phthisical patients against the swallowing of their expectorations, and advising the use of antiseptic mouth- and tooth-washes. A further lesson in prophylaxis consists in the avoidance of tuberculous meat and milk. Penzoldt advises, in case tuberculous sputum has been swallowed into a jejune stomach, that the patient should at once eat a little bread, because then free HCl develops sooner under the stimulus of the food and may destroy the vitality of the bacilli. Personally I prefer to advise all my tuberculous patients to wash their stomach out in the morning before breakfast with a solution of HCl 6 : 1000, combining this with the use of a carbolated mouthwash.

Histology of the Tuberculous Intestinal Ulcer.—The first visible beginning of tuberculosis of the intestines consists in altered lymph-nodules, which show themselves as gray, prominent lumps, which later become opaque and yellowish white. The solitary follicles swell by cell proliferation. In the center of these caseation gradually supervenes, the follicle breaks down and degenerates, and a crater-like ulcer develops. It is rare to find all or a great many of the solitary follicles or agminated glands involved together—generally only one follicle or gland is thus affected. When a Peyer's patch has been invaded by several primary ulcers, these become closely approximated in their extension, and thus the so-called confluent intestinal tubercular ulcers are developed. This large type of confluent ulcer retains its irregular shape, and may often develop a girdle shape, encircling the lumen of the intestine. The fact that in tuberculosis only single individual follicles of the plaque are attacked, and others of the same plaque may be comparatively free of the infection, constitutes a distinguishing feature histologically from the manner in which the typhoid infection involves this same glandular structure. In typhoid and simple catarrhal inflammation of the agminated follicles the entire plaque is uniformly diseased. The extension of the tubercular ulcer occurs, as a rule, in a direction at right angles to the long axis of the intestine. When a section of tubercular intestine is examined with a low power, not exceeding twenty diameters, the occurrence of numerous tiny nodules, which are found generally most abundantly in the neighborhood of ulcerations, arrests attention. The accompanying illustration illustrates a section through a tuberculous duodenal ulcer, and shows these nodules, which are small tubercles, very clearly. The central portion of these tuberculous granulations, particularly those near the edges of the ulcer, are frequently found converted into a caseous mass of debris which is unstainable. Woodward (*l. c.*, p. 587) presents some beautiful illustrations of this characteristic of intestinal tuberculosis. In his drawing (*l. c.*, p. 587, Fig. 37) he has undoubtedly observed that giant cells do occur in these nodules. When this surpassing work was written, the tubercle bacillus was unknown, and therefore we find no mention of it in Woodward's article on histology of the tubercular ulceration of the intestine.

---

* I am at present engaged in examining, with greatest regard for detail, all available intestines of subjects dead from tuberculosis, and hope soon to publish the results.

The *diagnosis and treatment* of tuberculous intestinal ulcers will be considered in the general chapter, together with symptomatology of all types of ulcers involving the bowel.

From the hygienic and prophylactic standpoint it is important to emphasize that the meats of tuberculous beeves may produce primary tuberculosis of the intestines (Baumgarten, Bollinger, and Orth, *l. c.*). It appears that a direct injury or desquamation of the epithelium is not necessary to make possible the invasion of the bacilli into the mucosa. Tuberculosis that has been caused by the ingestion of tuberculous milk, meat, or sputa begins in the lymph-follicles, and is generally accompanied by tubercular formation in the mesenteric glands.

Tubercular Rectal Ulcers.—These ulcerations originate from tuberculous nodules by necrosis. They generally have a girdle-shaped configuration. As they are observed clinically by inspection of the rectum, the periphery of the girdle-shaped ulcer pre-

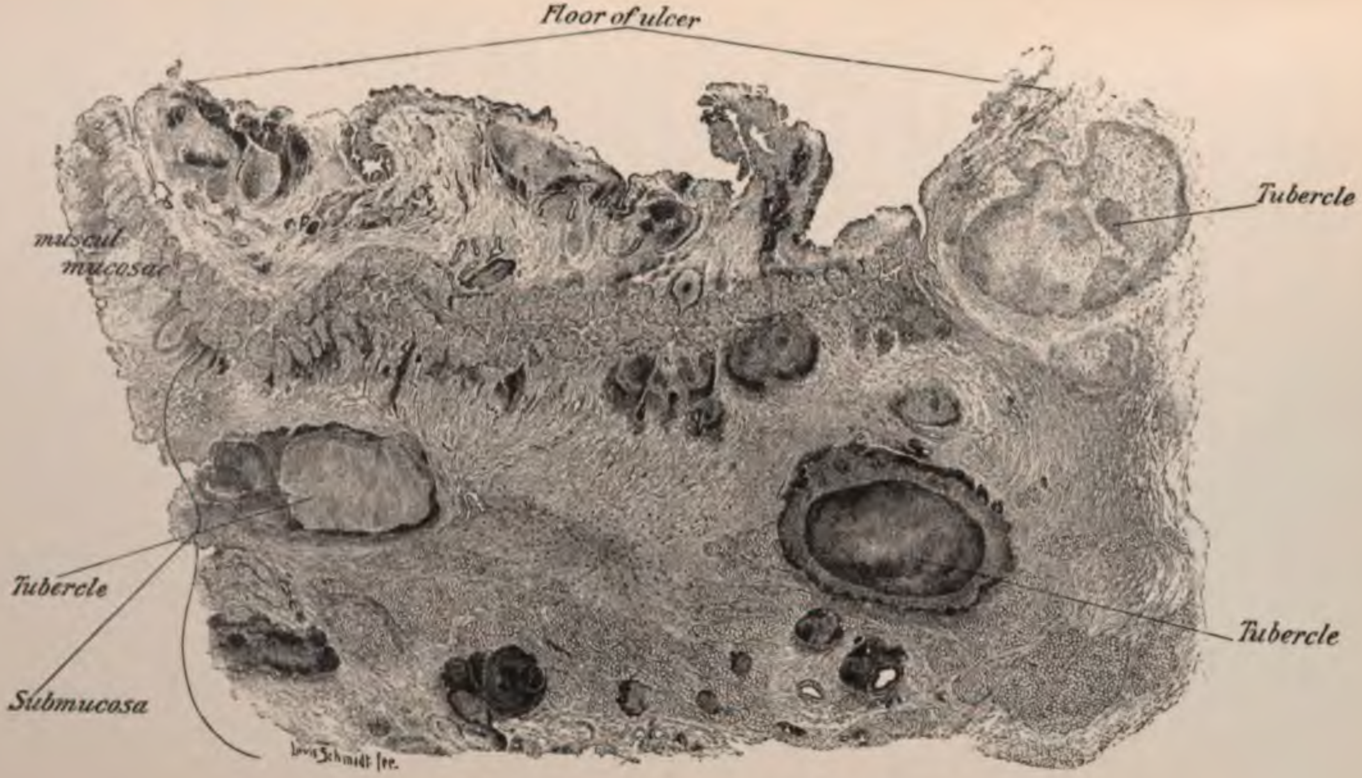


FIG. 35.—HISTOLOGY OF TUBERCULAR ULCER.

sents numerous smaller and younger tuberculous eruptions of a yellowish-gray color, which in turn undergo softening, caseation, and fatty degeneration, forming additional secondary tuberculous ulcers. They are frequent causes of periproctitis and rectal fistulæ. The tuberculous affections of the cecum—tuberculous perityphlitis and appendicitis—will be considered in another chapter. The diagnosis of tuberculous rectal ulcer depends upon the demonstration of the tubercle bacillus in the rectal excretions.

Gonorrheal Rectal Ulcers.—In women gonorrheal ulcers of the rectum are sometimes encountered which present diagnostic difficulties. This gonorrheal proctitis and periproctitis can be recognized whenever it is possible to demonstrate the presence of gonococci, which in the two cases of my experience it was not possible to do, although the ulcerations were undoubtedly gonorrheic. I was able to find only the ordinary pus cocci in the rectal discharges.

The diagnosis becomes difficult indeed when tuberculous individuals are subject to syphilitic infections, or vice versa. In these cases the diagnosis should be reserved until the results of antisyphilitic treatment can be noted or the effect of minute doses—two milligrams—of Koch's tuberculin can be observed. The detection of the tubercle bacillus establishes the diagnosis of tuberculosis in these cases, but it does not exclude the possibility of syphilis. There are rare cases of rectal ulcerations in which a carcinoma has developed on the basis of a syphilitic ulceration. Here an exact diagnosis of the original nature of the disease is almost impossible.

Syphilitic Intestinal Ulcers.

If we exclude what is known concerning syphilitic ulcerations of the rectum, our knowledge of syphilitic intestinal ulcers is very limited. I refer to what has been said concerning syphilitic ulcers of the stomach for the more detailed account of the histological process with which we are here concerned (Hemmeter, "Diseases of the Stomach," second edition, pp. 596 to 605). The observations concerning syphilitic infection of the intestine are perhaps more numerous than those made upon the stomach, but still quite scarce. A small number of anatomicopathological findings and a few clinical phenomena that have been studied during life comprise about all that is known concerning intestinal syphilis. These ulcerations have been observed in hereditary as well as in acquired syphilis, and occur in all sections of the intestine. In the duodenum they have been observed combined simultaneously with gastric ulcerations. Cases have been reported by Riedel (*l. c.*), E. Hahn (*l. c.*), Rieder (*l. c.*), Oser (*l. c.*), Aufrecht (*l. c.*), A. Beer (*l. c.*), A. Blackmore (*l. c.*), Förster (*l. c.*), Björnström (*l. c.*), Birch-Hirschfeld (*l. c.*), Hayem et Tissier (*l. c.*), Laurenzi (*l. c.*), Meschede (*l. c.*), Mracek (*l. c.*), Oestreich (*l. c.*), Simon (*l. c.*), Warfwinge und Blix (*l. c.*), Sorrentino (*l. c.*), and Norman (*l. c.*). Pathologically, syphilitic intestinal ulcers may be classed under five different headings, according to the manner in which they arise: (1) Specific intestinal ulcers which arise from ulceration of Peyer's patches and in rare instances of the solitary follicles, these changes representing, in fact, a syphilitic follicular enteritis, with consequent break-down of the histological elements most involved. The histological changes which the lymph-follicles undergo are quite as characteristic for syphilis as other corresponding changes are which have been found in the lymph-follicles in tuberculosis, enteric fever, and other diseases. (2) Specific ulcers of the intestine, brought about by softening and necrobiosis of circumscribed gummous infiltrations. (3) Intestinal ulcers brought about by ulceration of syphilitic amyloid degeneration of the intestinal mucosa. These three are due to a purely syphilitic infection. In addition to these there are two other rare forms of syphilitic ulcers which, according to Neumann, owe their origin to a mixed infection. These are: (4) Diphtheroid intestinal ulcers, which occur in the rectum in congenital and acquired syphilis, and form very extensive destruction of tissues. In a number of these types the ulcers undoubtedly have their origin from gummata, but in others they apparently do not arise from gummata, but, according to Neumann, are brought about by a peculiar histological process not yet satisfactorily investigated, and which he designates as (5) syphilitic malacia of the mucosa. Syphilitic ulcers of the small intestine are exceedingly rare in adults, but they are found oftener in children. Those that have been observed in the new-born assume the shape of gummatous infiltration of the mucosa and submucosa. In a few cases the syphilitic process was limited to the lymphatic apparatus. Miller (*l. c.*) has reported a case of syphilitic stricture of the jejunum at the place of transition into the ileum, where the lumen was so contracted as to be hardly passable by the little finger. The constricted portion was 1 ¼ inches long, and composed of fibrous cicatricial tissue, with total absence of the normal histological elements of the mucosa. The portions of the bowel above the constriction were dilated and showed a hypertrophy of the muscle layers.

In a negro who had suffered from syphilitic ulcers of the soft palate and syphilitic iritis, and who eventually succumbed to a status epilepticus in the hospital of the University of Maryland, gummata were found in the following places: one in the occipital lobe, one in the liver, one in the right kidney, and one in the left testicle. In the duodenum, just below the opening of the common gall-duct and involving the papilla of Vater, there was a circular ulcer extending nearly around the lumen of the gut; at its broadest portion it had a width of two centimeters. There were numerous small similar erosions in the ileum near its entrance into the cecum. There was also a syphilitic gastritis. The solitary follicles and Peyer's patches in various parts of the intestine were enlarged and prominent. The peritoneum corresponding to the ulcer in the duodenum was partially adherent with the parietal peritoneum and partially with the under surface of the liver. This negro was forty-two years of age. He confessed having had an attack of syphilis and had a scar on his penis. The ulcer in the duodenum had an infiltrated edge; the base was of a grayish-white color, irregular, and corroded.

Rieder's case was one of acquired luetic disease with twenty-two ulcers extending from the lower jejunum to the ileocecal valve. He could not prove to his own satisfaction that the ulcerations had arisen from Peyer's patches. They were, in his opinion, the result of degeneration of syphilitic neoplasms. From the examples of luetic ulcers of the bowels which are given in the literature on this subject it is evident that congenital as well as acquired syphilis may produce different pathological processes in the intestines. There are mainly two varieties: In the first we have to deal with a gummous enteritis in which the solitary and agminated follicles, even those quite close to the infiltration, are quite normal, but the mucosa is ulcerated and the submucosa very much thickened. The remaining layers of the intestinal walls are more or less involved, but in rare cases they may be unchanged. The second variety of cases are those in which the syphilitic process has its seat in the glandular patches of Peyer. We have already mentioned three other methods of origin, but they are exceptional. Luetic ulcers occur more often in the colon than in the small intestine, but the rectum is the most important location for this specific lesion from the clinical standpoint; here they occur most frequently, and here they are generally limited, the majority of cases of luetic rectal ulcers not being associated with similar lesions in other parts of the intestine. The most frequent location of these ulcers is from five to ten centimeters above the anus. They are distinguished from dysenteric ulcers by their shallowness, the much smoother character of their bases, and the relatively considerable cicatricial induration of their surroundings. Luetic rectal ulcers may originate in a variety of manners. In the first place, there is good reason for believing that they may be the result of break-down, of tertiary syphilitic neoplasms, or gummata; and, secondly, they may result from primary chancres of the rectum. Muron (*l. c.*) and others attribute specific strictures of the rectum to the conversion of broad condylomata into cicatricial tissue. Baeumler (*l. c.*) has called attention to the fact that these rectal strictures are most frequent in women between their seventeenth and thirtieth years, and Erskine Mason[1] has observed several cases of venereal rectal strictures in which there were no symptoms of constitutional syphilis, and in two cases the strictures preceded the development of these symptoms. I desire especially at this place to call attention to the oldest description of syphilitic affections of the rectum in the English language, and perhaps the oldest in any language. It is by Henry James Johnson, and was published in the "London Medical and Chirurgical Review," 1835 ("Observations on Ulcerations of the Rectum that Occur in Association with Condylomata and other Symptoms of Secondary Syphilis"). Syphilitic luetic ulcers of the rectum may be brought on chiefly in three different ways: (1) By syphilitic and mechanical lesions of the rectal mucosa caused by coitus per anum. These are rare. (2) The majority of luetic diseases of the rectum are consecutive affections brought on by continuity or contiguity. (3) The rectum may become affected independently of any syphilitic affection of the anus, vagina, or the peri- and pararectal connective tissue or of adjacent organs. Nickel and Poelchen (*l. c.*) have emphasized that ulcerations of the rectum which have been attributed to syphilis frequently have a different origin. As a general rule, strictures of the rectum are attributed to the cicatrization of luetic ulcers, but these authors, although they admit the greater prevalence of females among those afflicted with constricting rectal ulcers (among 219 such patients 190 were women), assign a portion of the rectal strictures which are ordinarily classed as syphilitic to gonorrheal infection. They explain that gonorrheal abscesses of Bartholin's glands may cause an invasion of the rectum by an extension of the suppuration of the glands situated between the vagina and the rectal submucosa. Nickel (*l. c.*), in accepting Poelchen's conception, suggests that ulcerations supposed to be syphilitic may have been caused by enemata or other directly traumatic insults to the rectal mucosa. The prevalence of females among the cases of luetic rectal strictures is largely explainable by their greater liability to infection through natural and unnatural sexual relation, and also by the closer anatomical relationship between the genitalia and the rectum in the female.

---

[1] Cited after Nothnagel, *l. c.*

ULCERATIONS OCCURRING IN CONSTITUTIONAL DISEASES—GOUT.

The rare cases of intestinal ulcers which have been reported as occurring in association with **gout** have been described in such a defective manner and are so devoid of objective pathological evidence that they may be passed over by simply referring to them. Nothnagel (*l. c.*) and Boas (*l. c.*) assert that they have not observed such cases. Hayem claims to have observed intestinal villi filled with concretions of uric acid (see Hans Herz, *l. c.*, p. 47) which may be conceived to injure the tissue to such an extent as to lead to the formation of an ulcer. Dieulafoy (*l. c.*) also describes an intestinal lithiasis as an expression of gouty diathesis. The relation, however, to the formation of intestinal ulcers, if any, is obscure.

Scurvy.

Inflammation and ulceration of the agminated follicles have been observed in association with scurvy. At the autopsy hemorrhagic spots and small erosions were found in places where the follicles had ulcerated. In this connection I wish to emphasize that the intestinal hemorrhages which occur in scurvy may be the result of a coexisting ulcerative colitis or dysentery (Litten, "Die hæmorrhagischen Diathesen," Nothnagel's "Spec. Path. u. Ther.," Bd. VIII, Th. 3).

Leukemia.

In leukemia intestinal ulcers have also been recorded. They are more frequently observed in the rare form of acute leukemia. Askanazy (*l. c.*), Fränkel (*l. c.*), Ebstein (*l. c.*), Heubner (*l. c.*), and Lauenstein (*l. c.*) have described such lesions in which the lymphatic structures of the intestine had become infiltrated and ulcers had resulted from secondary necrosis of these structures. Heubner mentions a hemorrhagic diphtheric colitis of an ulcerative type occurring in leukemia.

TOXIC ULCERATIONS.

Strictly speaking, all ulcers of the intestine which are caused by the corrosive action of irritant poisons that are taken directly into the alimentary canal should be classed under this heading. A great many vegetable and mineral poisons have been claimed to produce this effect,—for instance, the inorganic acids, phosphorus, arsenic, and mercury,—though probably the chief effect of these substances is exerted in the stomach and esophagus. The ulcerations that occur during typhoid fever, yellow fever, grip (Jürgens and Weichselbaum), variola, dysentery, cholera, anthrax, tuberculosis, actinomycosis, syphilis, and septicemia may also be classified as toxic or toxic infectious ulcers. Here I wish, however, to speak of the productions of toxic ulcers where the poison exerts its irritating action, as it were, by a constitutional route. Treitz (*l. c.*) has observed 19 cases of croupous and ulcerative dysentery and 12 cases of scab formations in the intestine in 220 cases of nephritis. The scab incrustations are described as having been partly fresh and partly gangrenous. The intestine constitutes an important path of elimination for systemic toxic substances, and there is no doubt that the detrimental compounds that are formed in the blood under the influence of a nephritis may give rise to ulcerative processes similarly as they cause diarrheas and intestinal catarrhs. It is probable that the urea which is excreted through the intestinal lumen is converted into carbonate of ammonia during the passage, and that it is this salt which exerts the detrimental irritation. Dickinson (*l. c.*) suggested that the albuminuric ulceration and follicular inflammations occurred from minute hemorrhages. The intestinal alterations that have been observed associated with Bright's disease are mainly of two kinds: The first and most frequent class of changes are those that run their course under the pathological picture of a diphtheric enteritis and colitis. This process may be circumscribed or very extensive, and lead to superficial as well as penetrating destruction of the intestinal wall. Occasionally the disintegration may assume a gangrenous character. The second are the typical ulcerations which occur in the solitary follicles and Peyer's patches. These ulcers vary considerably in size and occur in the ileum, colon, and rectum. The smaller ones have sharply defined and undermined edges. When several smaller ones coalesce, a larger ulcer is formed with irregular edges. Such ulcers have already been observed by Frerichs (*l. c.*) in the sigmoid flexure and the rectum.

Mercurial Ulcers.

In the chapter on Dysentery I have already sufficiently dwelt upon the rôle of mercury and its salt in producing intestinal ulcerations, which may occur in the small intestine, but more particularly in the colon. These toxic ulcers generally run their course under the clinical picture of a dysentery. When they occur in the rectum and sigmoid, they lose their individuality. I have seen a rectal ulcer in a male patient, aged twenty-four years, who had been treated for syphilis. The man was a Hungarian and could not speak the English language. It could not, therefore, be determined whether he admitted syphilitic infection or not. The inunction had been carried to the extent as to cause ptyalism, under which treatment the rectal ulcer became aggravated. After a discontinuation of the mercury and the administration of tincture of chlorid of iron the patient gradually improved, and the rectal ulcer was in a state of healthy granulation about three weeks after the iron treatment. There is no doubt in my mind that the patient was not syphilitic, and that the ulcer had been aggravated, if not caused, by the mercurial inunctions.

SYMPTOMATOLOGY AND DIAGNOSIS.

Whenever intestinal ulcers give rise to symptoms at all they will present themselves as diarrhea, pain, and blood, pus, mucus, tissue fragments, and characteristic bacteria in the stools. The majority of cases of intestinal ulcers which I have had occasion to observe clinically, and also at the autopsy, have run their course without giving rise to symptoms. When symptoms do develop, they vary according to the nature of the ulcerative processes and their localization and extent. The symptomatology of simple duodenal ulcer has already been fully described; also the clinical history of those which occur in association with cutaneous burns, and those which develop during the course of the various forms of dysentery. Outside of these the most frequent kinds of ulcer that will be observed by the general practitioner are the simple catarrhal, the tuberculous, and the follicular ulcers. In all cases in which there is the least suspicion of the existence of intestinal ulcers the rectum should be inspected by the methods given by Dr. Thomas Charles Martin in the section of Diseases of the Rectum. In my experience I have occasionally discovered an ulcer in the rectum and sigmoid when there were no symptoms or signs whatever pointing to this locality.

Diarrhea.—The more frequent and liquid stools which may occur are the result of an increased peristalsis effected by the irritation of the exposed nerve fibrils and the impaired absorption of water. In ulcers that occur in the upper part of the bowel, in the small intestine, and in the ascending colon, diarrhea is, as a rule, not observed. As the colon in its lower path—the transverse and descending colon—and the sigmoid constitute the main inhibition to the downward progress of the intestinal contents, one would suppose that ulcerative irritation of the nerves of these parts would inevitably cause diarrhea. As a matter of fact, this is occasionally the case, but there are many instances of chronic ulcers of the colon which are not associated with diarrhea, very likely because the long-standing and frequent irritation of a chronic ulcer is not followed by reaction. As a rule, all ulcer formations in the intestine are associated with an intestinal catarrh more or less intense, and it is this enteritis or colitis which gives rise to the symptoms of constipation, diarrhea, or mucus in the stools, as the case may be; although ulcers may be present in the colon, the accompanying chronic colitis may have dulled the irritability of the nervous apparatus and rendered it less responsive to the irritation of the ulcer. Experience has taught that the absorption of water also is quite normal so long as the greater part of the colon is intact. From the considerations in the chapter devoted to Peristalsis of the Intestine we know that an irritation exerted in any special locality of the intestine does not necessarily travel the entire extent of the bowel. Even irritations that strike the colon do not necessarily produce evacuation. Personally I have records of catarrhal and tuberculous ulcers in the cecum and ascending colon where the clinical history gave no evidence of diarrhea. Kortum (*l. c.*) has reported a case of almost entire destruction of the mucous membrane of the colon in a tuberculous woman, only a few islands of mucosa remaining intact. Before she was received into the hospital she suffered from diarrhea, which continued for nine days after her admission; but during the five weeks following and immediately preceding her death she had but one passage a day. Frerichs (*l. c.*) has reported two cases of extensive intestinal tuberculosis. In one there was profuse diarrhea, and the diagnosis of typhoid fever had been made. In the other there was obstinate constipation. This case had extensive tuberculous ulcers in the colon. Nothnagel assumes, in explanation of such cases, that the destructive process has either destroyed the nerves altogether in the region of the ulcers or that the nerves have lost their irritability as a result of the constant repetition of the stimuli. From all this it is evident that diarrhea in association with intestinal ulcers is a *very variable symptom. It may be present, but not necessarily so.* The factors that control the diarrhea are: (1) Location of the ulcer; ulcers high up in the small intestine are not so likely to produce diarrhea as those in the colon, cecum, and rectum; (2) the extent of the accompanying enteritis or colitis; (3) the absorptive power of the intestinal mucosa for water; and (4) the degree of irritability of the sensory nerves.

The Presence of Blood in the Stool.—The significance of blood has already been considered in the sections devoted to dysentery and simple duodenal ulcer. Blood may be present in a large variety of diseases other than ulcers: for instance, in states of passive congestion associated with hepatic cirrhosis, after traumatic influences; in association with hemorrhoids, neoplasms, enteritis, purpura, embolism of the mesenteric arteries, etc.

The blood, when it does occur in the stool in intestinal ulcers, may be found intimately mixed with the dejections or constituting a separate part thereof. If the source of the blood is high up in the intestine, we may not find blood as such, but only the decomposition products of blood. Often it is only demonstrable by the microscope or by chemical tests. The demonstration of hematin crystals in the evacuations is of doubtful value, since they may be derived from the meat contained in the food. Catarrhal and tuberculous ulcers do not give rise to the bloody admixtures of the stool so often as dysenteric and typhoid ulcers. Small hemorrhages may occur from ectatic veins of the rectum that have not yet developed into visible hemorrhoids. Here an inspection of the rectum with the speculum will prevent diagnostic errors. I have mentioned a number of diseases in the preceding which can give rise to blood in the stools; fortunately the accompanying clinical phenomena of these diseases are so characteristic and their etiological relations generally so well understood that a confusion with intestinal ulcers can occur but rarely. I may formulate the deductions from the foregoing considerations based upon my personal experience as follows: The absence of blood from the stool does not contraindicate the existence of intestinal ulcers, but if intestinal hemorrhages occur in association with well-recognized etiological factors of intestinal ulcers, then the presence of blood is a significant sign pointing with great probability to their existence.

The Occurrence of Pus.—Pure pus, meaning the aggregation of closely packed necrotic round cells, is rarely found in the dejections. Small amounts of pus can be detected only by the microscope, but frequently ocular inspection is sufficient to recognize its presence. While absence of pus is no contraindication to the existence of intestinal ulcers, I regard the presence of pus in the stool as one of the most important signs of ulceration that we have. To draw intestinal abscess and dysentery, and even necrobiotic neoplasms, into the consideration of the differential diagnosis serves rather more a theoretical than a practical purpose, because all these conditions are, after all, intestinal ulcerations from the clinical standpoint, and to have made this diagnosis would not be an error even if it should be proved to be one of the conditions mentioned. Abscesses, suppurating carcinomata, and dysenteric ulcerations give rise to larger quantities of pus; whereas the other forms of ulcer give rise only to small quantities. If we find little clumps of closely packed pus-cells microscopically in the stool, the diagnosis of ulcer is as good as confirmed, especially when one or other of the symptoms already mentioned are present.

Mixture of Pus, Blood, and Mucus.—Pure pus occurs only in perforating abscesses, and occasionally in the diphtheric dysenteries. Admixture of blood, pus, and mucus occurs in ulcerating carcinomata and catarrhal dysentery, especially when they are localized in the rectum and sigmoid.

Tissue Fragments.—In examining the evacuations for fragments of intestinal tissue it is always necessary to put the patient upon an exclusive milk diet for two or three days; otherwise the tissues can not be distinguished from the animal fibers taken in in the diet. When found, after such precautions, the presence of tissue fragments is a very important diagnostic indication. Unfortunately, they are found very rarely under such conditions. The diseases in which such fragments are found most frequently are the various forms of dysentery. In all other forms of ulcer they are searched for in vain in the evacuations.

Tubercle Bacilli.—The presence of tubercle bacilli does not furnish the diagnostic aid that was formerly attributed to them. Their presence is of importance only when we can secure the conviction that they come direct from the intestinal wall and are not derived from swallowed sputa. In rectal ulcers the material to be examined can be taken direct from the surface of the ulcer itself. Boclo (*l. c.*) found tubercle bacilli in the dejecta of three cadavers afflicted with intestinal tuberculosis; in three other cadavers which had no intestinal affection he found them also. In three further cases of intestinal tuberculosis his examination for tubercle bacilli in the dejecta was positive. He concluded that neither the negative nor the positive result of this examination for tubercle bacilli permitted of any conclusion for the presence or absence of intestinal tuberculosis. Notwithstanding these studies of Boclo, I feel justified in making the following deduction. When the tubercle bacilli are constantly found in great abundance in cases where there is no expectoration or sputa, we are justified in presuming that they are derived from the intestine.

Mucoid Masses Resembling Sago Granules.—Among the ingredients of the stools to which considerable interest has been attached are little transparent masses which have been compared to grains of boiled sago or frog spawn. They have been supposed to originate in the ulcerated solitary follicles of the colon, and therefore to afford clinical evidence of the existence of follicular ulcerations. At least this view has been held by such prominent clinicians as Bamberger, Finger, Griesinger, and Heubner. Virchow, however, calls attention to the fact that if these bodies are treated with dilute iodin, they acquire a blue color, and he remarked ironically that what had been called "sago-like mucus" is much more frequently "mucus-like sago." There can be no doubt that these grains resembling boiled sago are of vegetable origin. Frerichs ("Die Verdauung," Wagner's "Handwörterbuch der Physiol.," Bd. III, Abth. 1, S. 804) called attention to an observation according to which the starchy matters of the food are transformed into a tough, stringy, semitransparent substance very similar to mucus under certain abnormal conditions of the digestive processes, and that the same transformation can be imitated outside of the body by inducing lactic acid fermentation in fluids rich in starchy matters. I have already spoken of these bodies in the chapter on Dysentery and to the errors they have given rise to. So far as the diagnosis is concerned, I may say that these round mucoid globules have no significance whatever for the diagnosis of ulcerations. Ulcers do not secrete mucus, but form pus. Nothnagel (*l. c.*), Kelsch (*l. c.*), and Woodward (*l. c.*) agree in this view of the matter, so that the time-honored diagnostic importance of the round globules of mucus must be given up so far as intestinal ulcers are concerned. It is always better to place a patient on a strict diet of known composition in examining stools. If the diet has not contained any food of vegetable origin for several days, there will be less danger of confusing mucus with products of abnormal starch digestion. The composition of these "intestinal test-meals" will vary according to the object we wish to accomplish (see Chap. IX). For the search for blood, mucus, bile, etc., a milk diet is preferable.

Pain.—This is the only subjective symptom that occurs in association with intestinal ulcers. Pain may be absent entirely, though the ulcers can later on be demonstrated at the autopsy. This is especially the case in chronic ulcers. The symptom of pain has some diagnostic significance when it is very circumscribed, always limited to the same spot, and is increased on pressure. There are mainly two kinds of pains that are observable in this connection: First, the localized pains, not quite so intense; and, secondly, the diffuse and colic-like pains. Sometimes they are not noticeable nor complained of by the patient except after pressure on the abdomen. According to Leube and Nothnagel, the pains are more frequently due to a mechanical irritation of the circumscribed peritonitis which generally accompanies a chronic ulcer, than to direct irritation of the ulcer itself. Of course, it is not denied that direct pressure over an ulcer can also cause pain, but it is emphasized that the peritoneum, where it is involved in the inflammation or ulcerative process, is the principal cause of the pain. Only in two localities can the pain of intestinal ulcer be said to be characteristic—in the beginning and end of the intestine. In the duodenum ulcers cause cardialgia, and in the rectum they cause tenesmus. In my work on "Diseases of the Stomach" I have emphasized that even gastric ulcers may run their course without pain. It is, therefore, not a matter of surprise that intestinal ulcers, which are much less subject to irritation by HCl and are not subject to such frequent compression by the peristalsis from the part in which they are located, should run their course without pain. In the differential diagnosis between enteritis and ulceration the constant presence of a localized pain on pressure should be interpreted as signifying an ulceration. Ulcerations when penetrating through the intestinal wall will cause a circumscribed peritonitis presenting the symptoms of increased pain and a limited dullness on percussion, caused by the exudate and the abscess formation. General peritonitis from this cause is connected with perforation of the intestine and extravasation of air into the peritoneal cavity. As there are portions of the intestine which are devoid of the serous covering,—for instance, the posterior part of the descending, transverse, and inferior duodenum, and also of the ascending and descending colon,—ulcers may perforate in these localities without involving the peritoneum. Under such conditions the perforating ulcer is liable to set up purulent inflammation of the cellular tissue in the neighborhood of the kidneys. The pain on pressure over the colon—particularly the descending colon and sigmoid flexure—which can be elicited in cases of chronic dysentery has been considered in the chapter devoted to this disease. Pain that is not localized can not be available for the diagnosis of ulcer. Of the remaining symptoms, the *meteorism*, which is most frequent in the various forms of dysentery and intestinal tuberculosis; the *fever*, which is more a concomitant of the fundamental disease, such as tuberculosis and dysentery; or of the ensuing *peritonitis*, I can only say that they present nothing characteristic. Intestinal abscesses and suppurating carcinomata may give rise to very high temperatures, but the catarrhal and follicular ulcers in themselves are not accompanied by fever.

Concerning the ulcer formation in the vermiform process of the appendix, I will speak more fully in the chapter on Appendicitis.

The Urine.—Chvostek and Stromayer (" Wien. klin. Wochenschr.," Bd. XCVI, No. 47) have experimentally demonstrated alimentary albumosuria in cases of tubercular ulcerations of the intestines; after administering larger quantities of dry peptone or somatose they could demonstrate albumoses in the urine after a few hours in cases where there were tubercular ulcers of the intestine. Whether this observation, as well as the Ehrlich diazo reaction, can be utilized for the diagnosis of these ulcers can not be decided until more experimental clinical evidence on this subject has been collated.

The Condition of General Nutrition.—This is influenced more by the underlying disease than by the ulcers themselves. Small and isolated ulcers can not injure the general nutrition, but if diarrhea and destruction of the absorptive surface are caused by extensive and severe ulcerations, then great emaciation must be the inevitable result.

Critical Consideration of Diagnostic Factors.

From all that has been said on symptomatology and diagnosis it is evident that the diagnosis of intestinal ulcers can rarely be made with a precision that would be desirable. The only reliable sign of intestinal ulcerations that we have is the presence of pus and tissue fragments in the stool; the liquid condition and the number of the evacuations permit of no conclusions. The pain is of very limited diagnostic value—only then when it occurs in the same locality on pressure. Strictly circumscribed pain on pressure, associated with fever and preceded by a clinical history suggestive of ulcer, speaks in favor of a perforation peritonitis. The presence of blood in the evacuations in association with the other well-recognized etiological factors of intestinal ulcers is a sign that is available for the diagnosis. The determination of the disease is, of course, impossible where it runs its course without symptoms; frequently the diagnosis can not be made even when there are symptoms, and the clinician will have to await the results of treatment before he can make up his mind. This is particularly the case in differentiation of tubercular, amyloid, and syphilitic ulcers. The result of antisyphilitic treatment, on the one hand, and of treatment for tuberculous ulcer, on the other, may shed some light on the subject. Petruschky ("Verhandl. d. Cong. f. innere Med.," 1899, p. 366) has reported two cases of tuberculous ulcer of the stomach which were apparently cured by injections of tuberculin. Typical reactions which followed the injections of this substance established the diagnosis of these ulcers, which had been considered simple peptic ulcers prior to the injection (see Hemmeter, "Diseases of the Stomach," second edition, pp. 515 and 594). I have repeated this method of diagnosis by injecting one milligram of tuberculin in a male patient suffering from an ulcer of the rectum that could be distinctly seen on the anterior wall of this organ. The lungs were normal to auscultation and percussion. The first injection of one milligram gave no distinct reaction. Two days thereafter I injected five milligrams, which was followed by a fever of 103.5° F. and great pain, with tenesmus in the rectum. The ulcer, which had up to that time been of a grayish-white color with corroded irregular base, assumed a dry, highly red, and more inflamed appearance. Up to the sixth injection increased sensitiveness of the rectum and colon was very evident. After that the rectal pain became less. Two weeks after the first injection the ulcer was undoubtedly healing. I increased the dose to fifty milligrams, which was given three times a week. At the end of the fifth week the sore had completely healed. I did not measure the ulcer, but should say that it was about the size of a five-cent piece, with a smaller ulceration about the size of a split pea to the right side of the main loss of substance. The patient had a tubercular history, the mother and a sister having died from pulmonary tuberculosis. He was examined fourteen months after the first injection of tuberculin and no sign of rectal ulceration could be discovered. The local treatment consisted of colon irrigations with sterile normal salt solution; no internal medication was given, but a highly nutritious diet of milk, soft-boiled eggs, nutrose, and scraped beef.

On the basis of this experience I am inclined to hope that in tuberculin we may have a means of establishing the nature of tubercular ulcers of the rectum, at least where this is doubtful.

PROGNOSIS.

The prognosis of intestinal ulcer depends upon the etiological nature of the underlying disease and upon the extent of the destruction of the intestinal mucosa. The locality has some influence on the prognosis, but it is not very far-reaching. Some forms of ulcer heal very readily—for instance, the typhoid, dysenteric, and syphilitic ulcers; others show very little tendency toward healing; such are the tuberculous, embolic, and thrombotic ulcers; still others never heal; among these I should class the amyloid and carcinomatous ulcers.

TREATMENT.

The therapeutics of the duodenal and dysenteric ulcers have already been considered in separate chapters. Syphilitic ulcers, whenever the diagnosis can be established, naturally call for antisyphilitic treatment. All other ulcerations I can safely say will not be diagnosed except the catarrhal and tuberculous ulcers. Naturally, where the ulcers occur in the rectum they can be recognized by inspection. So the object of the treatment can be narrowed down practically to the management of the pain, the intestinal hemorrhage, and diarrhea, and the direct treatment against the ulcerative process itself. Unfortunately, our means of treatment of intestinal ulcerations is very limited. They are restricted to checking the diarrhea, preventing further injurious influences from reaching the intestine, and relieving pain. The diarrhea is best controlled by the diet.

Diet.

This has already been considered in a separate chapter. The patient should be confined to bed, and the following diet administered:

DIET FOR DIARRHEA IN ASSOCIATION WITH ULCERS.

8 A.M.—1 cup of chocolate—preferably acorn chocolate—boiled with very thin milk or water.
1 Zwieback or toast and butter.

10 A.M.—1 cup strained oatmeal gruel, or beef bouillon or chicken broth with rice.
50 gm. of baked blue fish, trout, or four raw oysters.

1 P.M.—Soup made of peas or beans, with bouillon and egg added; eucasin, tropon, or nutrose may be added to heighten the nutritive value of the soup; of vegetables, only potato in purée form; of meats, only calf's-brain, sweetbreads, and tender fish, without many bones, are permitted. All fruits are forbidden, with the exception of strained huckleberries, which can be added to gelatin.
For desserts, egg-custard, gelatins flavored with claret and a minimum amount of lemon-juice.
Beverages, old Burgundy and claret, and Rockbridge alum water—not over six ounces at a time of the latter.

4 P.M.—1 cup of tea, sweetened with saccharin, cakes, toast, or Zwieback, with butter.

7 P.M.—Meat pulp, very finely scraped in the kitchen at home (never to be bought already ground or chopped up by the butcher), seasoned with salt and rapidly broiled in butter.
1 glass huckleberry wine, strained, or good claret.
Stale wheat bread or toast with butter.

Whenever experience has taught that milk is well digested, it should be allowed a liberal place in the diet-list, but where it causes diarrhea it must be strictly forbidden.

Treatment of Pain.

Where it exists, this will be remedied by the treatment directed toward the ulcer itself—for example, the astringent preparations, bismuth salts, betanaphthol bismuth subgallate, and the antiseptic colon irrigations often arrest the pain of intestinal ulcers, but where the pain continues in spite of these means, the use of opium is unavoidable, especially when the diarrhea is troublesome; but it is rarely sufficiently intense to call for the hypodermic use of morphin.

The **treatment of hemorrhage** has been given in chapter xxvii. on Enterorrhagia. Lead acetate, bismuth subnitrate and salicylate, and nitrate of silver are of doubtful value in the treatment of this symptom. In the treatment of ulcers lying high up in the intestine I have obtained the most satisfactory results obtained under any treatment by the use of bismuth subnitrate fifteen grains five times daily, together with $\frac{1}{6}$ of a grain of the denarcotized extract of opium. The bismuth is supposed to form a protective covering for the surface of the ulcer. This is a hypothesis, for in two cases of tubercular ulcer that had been taking bismuth subnitrate for months I found at the autopsy that this chemical was anywhere and everywhere except on the ulcers. Nevertheless this treatment gives comfort to the patients in most cases where the ulcer cannot be treated locally. Alum, nitrate of silver, the styptic iron preparations, and tannin have, in my experience, done considerable damage by injuring the gastric digestion, while they did not exert any evident beneficial effect on the intestinal ulceration.

Direct Local Treatment of the Ulcerative Process.

This is possible only where the ulcers are located in the colon, sigmoid, or rectum. Those located in these sections should be treated by the direct methods described by Dr. Thomas Charles Martin in the chapter on Diseases of the Rectum. Ulcers in the colon can be more successfully treated than those higher up in the bowel, because they can be reached by high colon irrigations. In order that these should be successful, however, we must first check the diarrhea, for one or, at the highest, two irrigations a day can exert no very decided astringent or healing effect on the ulcers if it is immediately recovered by from five to ten evacuations in twenty-four hours. Rest in bed, diet, opium, and bismuth subgallate by the mouth, with hot applications externally on the abdomen, will, with very few exceptions, reduce the evacuations to the normal number; then, if the cause of the diarrhea should be in the colon, they will be further checked by the colon irrigations which can be now undertaken. The method has already been described in the chapter on Enteritis and also in that on Dysentery. The disinfecting and astringent substances with which I have experience, and which can be safely used, are thymol 1 : 2000, salicylic acid 1 : 300, boric acid

1 : 500, nitrate of silver 0.2 per cent., and tannin or tannigen 0.2 to 0.5 per cent. Very good results have, in my experience, followed irrigation with suspensions of equal parts of bismuth subgallate and bismuth subnitrate, similar to the irrigations recommended by Fleiner for gastric ulcers: a heaping teaspoonful of each of these bismuth salts is stirred into one liter (or one quart) of warm normal salt solution. The patient can, by gradual habituation, retain this irrigation for from fifteen to twenty minutes. It constitutes one of the few gratifying features in the therapeutics of intestinal ulcers.

I have personally observed the healing of a tubercular rectal ulcer under the administration of Koch's tuberculin hypodermically and locally.

LITERATURE ON INTESTINAL ULCERS

(Including Tuberculous, Syphilitic, and Other Ulcers Mentioned on p. 593).

1. Askanazy, “Ueber acute Leukämie und ihre Beziehungen zu geschwürigen Processen im Verdauungskanal,” “Virchow's Archiv,” Bd. cxxxvii.

2. Acland, “Pathological Transactions,” 1885.

3. Albers, “Die Darmgeschwüre,” 1831.

4. Aufrecht, “Amyloidgeschwüre des Darmkanals,” “Berlin. klin. Wochenschr.,” 1869, No. 30.

5. Alvazzi-Delfrate, “Gazz. med. di Tirino,” 1897, No. 7, cit. nach “Centralbl. f. innere Med.,” 1897, S. 845.

6. Behrens, “Ueber primäre tuberculöse Darminfektion des Menschen,” Inaug. Diss., München, 1891.

7. Baumgarten, “Die Histogenese des tuberculösen Processes,” Berlin, 85.

8. Boas, “Diagnostik und Therapie der Darmkrankheiten,” Leipzig, 1899.

9. Bokai, “Arch. f. exper. Path. u. Pharmakol.,” Bd. xxiii.

10. Boucquoy, “Arch. gén. de Méd.,” 1887.

11. Bardine, “Jour. of Exper. Med.,” vol. ii, p. 501.

12. Brambillo, cit. nach Virchow-Hirsch's “Jahresber.,” 1882, Bd. ii, S. 168.

13. Bayle, “Rech. sur la Phthisie Pulmonaire,” Paris, 1810, p. 59.

14. Bryant, “Semaine Med.,” 1893, p. 335.

15. Biedert, “D. Tub. d. Darms u. der lymphat. Apparate,” “Jahrb. f. Kinderheilkunde,” Bd. xxi, S. 158, 1884.

16. Burwinkel, “Deutsche med. Wochenschr.,” 1898, No. 52.

17. Birch-Hirschfeld, “Lehrbuch d. patholog. Anatomie,” Leipzig, 1887.

18. Billroth, cit. from Conrath, *l. c.*

19. Bäumler, Art. “Syphilis,” in von Ziemssen's “Handb. der spec. Path. u. Ther.,” Bd. iii.

20. Brissaud, cit. from Collin, *l. c.*

21. Billroth, “Ueber Duodenalgeschwüre bei Septikämie,” “Wien. med. Wochenschr.,” 1867, No. 45.

22. Bollinger, “Ueber Tuberkelbacillen im Euter einer tuberculösen Kuh,” “München. ärtzlich. Intelligenzblatt,” 1883, No. 16.

23. Baumgarten, "Ueber d. Uebertragbarkeit der Tub. durch die Nahrung," "Centralbl. f. klin. Med.," 1884.

24. Chvostek, "Medicinische Jahrbücher," Wien, 1883, H. 1, S. 1–58.

25. Chvostek, "Das Einfache Duodenal Geschwür," "Wien. med. Jahresb.," 1885.

26. Codivilla, "Sperimentale," Mem. orig., vol. XLVII, pp. 4 and 6.

27. Collin, "Étude sur l'ulcère simple du duodénum," "Thèse de Paris," 1894.

28. Collin, "Scot. Med. and Surg. Journal," July, 1897.

29. Conrath, Bruns' "Beiträge zur klin. Chirurgie," Bd. XXI, H. 1, 1898.

30. Czerny, Bruns' "Beiträge zur Chirurgie," Bd. VI u. IX.

31. Curling, "Med.-chir. Transactions," 1842, vol. XXV.

32. Colberg, "Beiträge zur normalen und patholog. Anatomie der Lungen," "Deutsch. Arch. f. klin. Med.," Bd. II, 1867.

33. Courtois-Suffit, "Maladies du péritoine," "Traité de Médecine," publié par Charcot, Bouchard, Brissaud, Paris, 1892, tome IV.

34. Curschmann, "Die Pocken," in von Ziemssen's "Handb. der spec. Path. u. Ther.," Bd. II, 2. Aufl., Leipzig, 1877.

35. Cohnheim, "Vorlesungen über allgemeine Pathologie," Berlin, 1880.

36. Darras, "De la perforation de l'ulcère simple du duodenum," "Thèse de Paris," 1896.

37. Devic und Roux, "Province médicale," 44, 46, 47, 1895.

38. Dickinson, "Roy. Med. and Chirurg. Society," January 9, 1894.

39. Dobroklonsky, "Archives de Medécine expérim.," 1890, No. 2.

40. Dickinson, "Transactions of the Pathological Society of London," vol. XVIII, 1866–67; vol. XXIX, 1879.

41. Dieulafoy, "Presse Medicale," 1895. "Lithiase intestinale et entérocôlite sableuse," "Cliniques de l'Hôtel-Dieu," 96, 97.

42. Eichhorst, Schmidt's "Jahrbücher," Bd. CCXX, S. 23.

43. Eisenhart, "Ueber Häufigkeit und Vorkommen der Darmtuberculose," Inaug. Diss., München, 1891.

44. Elbers, "Darmgeschwüre," 1831.

45. Ewald, "Berlin. klin. Wochenschr.," 1885, No. 32.

46. Ebstein, "Ueber die acute Leukämie und Pseudoleukämie," "Deutsch. Arch. f. klin. Med.," Bd. XLIV.

47. Eberth, "Virchow's Archiv," Bd. LXXX, 1880, S. 166.

48. Edinger, "Arch. f. klin. Med.," Bd. XXIX, S. 567, 1881.

49. Fenwick, Samuel and W. Soltau, "Ulcer of the Stomach and Duodenum," Philadelphia, 1900.

50. Fischer, "Arch. f. exper. Pathol.," Bd. XX, 1886.

51. Förster, "Handbuch der patholog. Anatomie," Leipzig, 1854.

52. Friedreich, "Einige Fälle von ausgedehnter amyloider Entartung," "Virchow's Archiv," Bd. XI.

53. Frerichs, "Die Bright'sche Nierenkrankheit," Braunschweig, 1851.

54. Frerichs, "Beit. z. Lehre von d. Tuberculose," 1882.

55. Girode, "Contribution à l'étude de l'intestin des tuberculeux," "Thèse de Paris, 1888.

56. Greenwood, "Lancet," August 21, 1880.

57. Giacomi, de, "Fortschr. d. Med.," Bd. I, No. 5.

58. Grawitz, "Statist. und exper. Beitrag zur Kenntniss der Peritonitis," "Charité Annalen," Bd. XI, 1886.

59. Gottsacker, "Zur Histogenese der tuberculösen Darmgeschwüre," Inaug. Diss., Bonn, 1880.

60. Hadham, "Lancet," February 18, 1871.

61. Hamann, "Statistik der Tuberculose im Alter von 16 bis 90 Jahren," Inaug. Diss., Kiel, 1890.

62. Hartmann and Pilliet, cited by Conrath, *l. c.*

63. Herz, Hans, "Die Störung d. Verdauungs-Apparates als Ursache u. Folge anderer Erkrankungen."

64. Herczl, "Orvosi Hetilap," 1895, No. 50; cit. nach "Arch. f. Verdauungs-Krankheiten," Bd. II, S. 251.

65. Herxheimer, "Deutsch. med. Wochenschr.," 1885, No. 52.

66. Höning, "Ueb. d. Auftreten d. Bacillen bei Darmtuberculose," Diss., Bonn, 1885.

67. Hofmeister, Bruns' "Beiträge zur Chirurgie," Bd. XVII, S. 577, 1896.

68. Houze, "De l'ulcère perforat du duodenum," "Thèse de Paris," 1896.

69. Hunter, W., "Pathological Transactions," 1890.

70. Heubner, Art. "Dysenterie," in von Ziemssen's "Handb. der spec. Path. und Ther.," Bd. II.

71. Heubner, see Hans Herz (*l. c.*), p. 531.

72. Illoway, "Constipation," New York, 1897, p. 136.

73. Klebs, "Pathologische Anatomie," 1869, Bd. I, S. 256.

74. Kinnicutt, Jacobi Festschrift, 1900.

75. König, "Deutsch. Zeitschr. f. Chir.," Bd. XXXIV, 1892, S. 65.

76. Körte, *ibid.*, Bd. XL, 1895, S. 523.

77. Kijanitzin, "Virchow's Archiv," Bd. CXXXI, 1893, S. 436.

78. Krauss, J., "Das perforirende Geschwür im Duodenum," Berlin, 1865.

79. Koranyi, Zoonosen, "Spec. Patj. u. Ther.," edited by Nothnagel, Bd. V, Th. 5.

80. Krokiewicz, A., "Wien. klin. Wochenschr.," 1898, No. 29.

81. Kussmaul und Maier, "Aneurysma verminosum hominis, Periarteritis nodosa," "Deutsch. Arch. f. klin. Med.," Bd. I, S. 125 u. 484, 1866.

82. Kortum, "Ueber Enterophtose," Inaug. Diss., Berlin, 1879.

83. Kelsch, "Archives de physiologie normale et pathologique," 1877.

84. Kerling, "Med.-chir. Transactions," 1842, vol. XXV.

85. Kitagawa, "Beiträge zur Diagnostik der Darmkrankheiten," Inaug. Diss., Würzburg, 1889.

86. Kyber, "Virchow's Archiv," Bd. LXXXI, S. 305, 1880.

87. Landau, L., "Ueber Meläna der Neugeborenen, nebst Bemerkungen über die Obliteration der fötalen Wege," Breslau, 1874.

88. Landerer und Glücksmann, "Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie," 1896, Bd. I, S. 168.

89. Lange, "Annals of Surgery," 1893, vol. II, p. 36.

90. Lennander, "Ueber Appendicitis," 1895, S. 29.

91. Lennander, "Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie," 1898, Bd. IV, H. 1, S. 91.

92. Lapponi, Virchow-Hirsch's "Jahresbericht," 1895.

93. Leichtenstern, "Deutsch. med. Wochenschr.," 1882, No. 58.

94. Leube, von, von Ziemssen's "Handbuch," Bd. VII, 2; 2. Aufl., 1878, S. 310.

95. Leube, von, "Specielle Diagnose innerer Krankheiten," 2. Aufl., S. 274.

96. Litten, "Zeitschr. f. klin. Med.," Bd. II, S. 702, 1881.

97. Lockwood, "Trans. Med. Soc. Lond.," 1895, No. 26, vol. XV, p. 91; ref. in "Centralbl. f. Chir.," 1895, No. 26.

98. Long, "London Med. Gaz.," N. S., vol. 1, p. 743.

99. Lorenz, H., "Beitrag zur Kenntniss der multiplen degenerativen Neuritis," "Zeitschr. f. klin. Med.," Bd. XVIII, H. 5 u. 6.

100. Larcher, "Archives de méd.," 1864 and 1865.

101. Lauenstein, "Zur Pathologie der Leukämie," "Deutsch. Arch. f. klin. Med.," Bd. XVIII.

102. Leonhardi-Aster (Birch-Hirschfeld), "D. Ztscjr. f. prakt. Med.," 1878, No. 8.

103. Mackenzie, "St. Thomas Hospital Report," 1892, vol. XX, p. 341.

104. Mayer, "Die Krankheiten des Zwölffingerdarmes," Düsseldorf, 1844.

105. Mayo, "Surgery of the Stomach," "Lancet," March 19, 1900.

106. Melchior, cit. nach Virchow-Hirsch's "Jahresbericht," 1890, Bd. I.

107. Meyer, Diss., Heidelberg, 1886.

108. Mering, von, "Verhandl. d. Cong. f. innere Med.," 1893.

109. Mikulicz, "Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie," 1897, Bd. II.

110. Minkowski, in Naunyn's "Mittheilungen aus der med. Klinik in Königsberg," S. 59, Leipzig, 1888.

111. Moritz, "Verhandl. d. 65. Versammlung der Gesellschaft deutscher Naturforscher und Aerzte in Nürnberg," 1893.

112. Meschede, "Zur Casuistik der Lungen- und Darmsyphilis," "Virchow's Archiv," Bd. XXXVII.

113. Muron, "Gazette méd. de Paris," 1873, No. 11.

114. Morot, "Thése de Paris," 1892.

115. Monti, "Centralzeitung f. Kinderheilkunde," Bd. II, 1879, No. 7.

116. Nothnagel, "Darmkrankheiten," "Spec. Path. u. Therapie," Bd. XVII.

117. Nickel, "Ueber die sogenannten syphilitischen Mastdarmgeschwüre," "Virchow's Archiv," Bd. CXXVII.

118. Obrastzow, "Arch. f. Verdauungs-Krankheiten," Bd. IV, 1898, S. 440.

119. Oppenheimer, "Das Ulcus pepticum duodenale," Inaug. Diss., Würzburg, 1891.

120. Orth, "Virchow's Archiv," Bd. LXXVI, 1879.

121. Oser, "Arch. f. Dermatol. und Syphilis," 1871.

122. Pagenstecher, "Die chir. Behandl. des Duodenalgeschwürs," "Deutsch. Zeitschr. f. Chir.," August, 1899.

123. Pölchen, "Zur Ætiologie der stricturirenden Mastdarmgeschwüre," "Virchow's Archiv," Bd. CXXVII.

124. Pauly, "Aerztliche Sachverständigen-Zeitung," 1897.

125. Perry and Shaw, "Guy's Hospital Reports," 1894, p. 171.

126. Parenski, "Wien. med. Jahrbücher," 1876, H. 3.

127. Ponfick, "Zur Casuistik der Embolie der Arteria mesenterica superior," "Virchow's Archiv," Bd. L.

128. Petitbien, "Des ulcérations intestinales dans l'érisipèle," "Thèse de Paris," 1883.

129. Rokitansky, "Allgemeine Abhandlungen über Darmtuberculose," Lehrb. III, 235, 1861.

130. Reckmann, "Ueber Ulcus duodenale und seine Diagnose," Inaug. Diss., Berlin, 1893.

131. Robin, Alb., cit. from Collin, *l. c.*

132. Rokitansky, "Ueber Darmeinschnürungen," "Oesterreichische med. Jahrbücher," Bd. X, 1836.

133. Rosenstein, "Die Pathologie und Therapie der Nierenkrankheiten," 2. Aufl., Berlin, 1870.

134. Spillmann, "De la Tuberculisation du tube digestiv," "Thèse d'aggrégation," Paris, 1878.

135. Salzer, "Von Langenbeck's Archiv," Bd. XLIII.

136. Schulze, "Beiträge zur Kenntniss des perforirenden Duodenalgeschwürs," Inaug. Diss., Greifswald, 1873.

137. Schrötter, "Aerztlicher Bericht des k. k. Allgemeinen Krankenhauses zu Wien," 1887, S. 27.

138. Scheild, "Internat. Magaz.," 1895, p. 866.

139. Starke, "Deutsche Klinik," 1870.

140. Schwartz, "Ulcère perforante du duodenum," "Bullet. et Mém. Soc. de Chirurg.," January, 1898.

141. Stich, "Deutsch. Arch. f. klin. Med.," 1874.

142. Steiner und Neureutter, "Prager Viertelj.," Bd. LXXXVI, S. 37, 1865.

143. Tschitschowisch, "Annales de l'Institut Pasteur," 3. Jahrg., No. 5, S. 222.

144. Treitz, "Prager Viertelj.," 1859.

145. Traube, "Gesammelte Beiträge zur Physiologie und Therapie," Bd. III, Berlin, 1878.

146. Virchow, "Ueber katarrhalische Geschwüre," "Berlin. klin. Wochenschr.," 1893, No. 8 u. 9.

147. Vonwyl, "Ulcer Perforans Duodeni," "Thèse de Paris," 1893.

148. Volkmann, "Arch. f. klin. Chir.," Bd. XXXII, S. 116, 1886.

149. Wagner, "Zur Diagnostik und Therapie der perforativen Peritonitis," "Deutsch. Arch. f. klin. Med.," Bd. XXXIX.

150. Wannach, "Arch. f. klin. Chir.," 1898, Bd. LVI, H. 2.

151. Wier, "New York Medical Record," vol. LVII, No. 18, May 5, 1900, p. 749.

152. Wittstock, "Zur Klinik des Ileus durch Darmtuberculose," Inaug. Diss., Berlin, 1893.

153. Wyss, "Correspondenzblatt f. Schweizer Aerzte," 1893, No. 22.

154. White-Hale, "On Simple Ulcerative Colitis," "Guy's Hospital Reports," tome XXX, 1890.

155. Widerhofer, von Gerhardt's "Handbuch d. Kinderheilkunde," Bd. IV, 2, S. 592, 1880.

156. Woodward, "The Med. and Surg. History of the War of the Rebellion," vol. I, part 2.

157. Wesener, "Kritische und experimentelle Beiträge zur Lehre der Fütterungstuberculose," Freiburg, 1885.

158. Wild, "Beiträge zur Kenntniss der amyl. und der hyalinen Degeneration des Bindegewebes," Diss., Würzburg, 1885.

159. Ziehl, Diss., Heidelberg, 1881.

160. Ziegler und Nauwerck, "Beiträge zur pathol. Anat. und Physiol.," Bd. I, S. 177, 1886.

CHAPTER XXII.

INTESTINAL NEOPLASMS.

I. MALIGNANT NEOPLASMS.—Carcinoma, Sarcoma, and Lymphosarcoma.

CANCER OF THE INTESTINES (CARCINOMA INTESTINALE), CANCER OF THE SMALL INTESTINE, OF THE LARGE INTESTINE, AND OF THE RECTUM.

Malignant neoplasms of the intestines exhibit analogous structural relations, both in external appearances as well as in their finer architecture, to the gastric malignant growths. It is important to bear in mind that the cylindrical-celled carcinoma is the type or fundamental form of all intestinal cancers. There are but two exceptions to this rule—one is a cancroid, or flat, squamous, epithelial-celled neoplasm occurring at the anus; the second is a glandular carcinoma that has its origin very probably in the duodenal Brunnerian glands. With these two exceptions the majority of intestinal cancers can be designated as "adenocarcinomata."

Existence, Extent, and Etiology.

Cancer of the intestines is observed much more rarely than cancer of the stomach, the uterus, and the mammary gland. Intestinal cancers are, in the majority of cases, primary diseases; more rarely, secondary. In the latter case the neoplasm is caused by the fact that a cancer of neighboring organs—stomach, omentum, lymphatic glands, bladder, prostate, uterus, ovary, and others—spreads to the intestines by contiguity or by disintegration of a cancer in other organs, whereby small cancer particles are carried to the intestine and there develop (metastasis). Favorite places for cancer are, in the first place, the rectum near the internal sphincter; in the large intestine, the flexures, the cecum, and vermiform process; in the small intestine, the neighborhood of the duodenal papilla, parts of the intestine which are more irritated than other parts by the acid chyme or stagnating fecal masses. According to a compilation of von Leube,

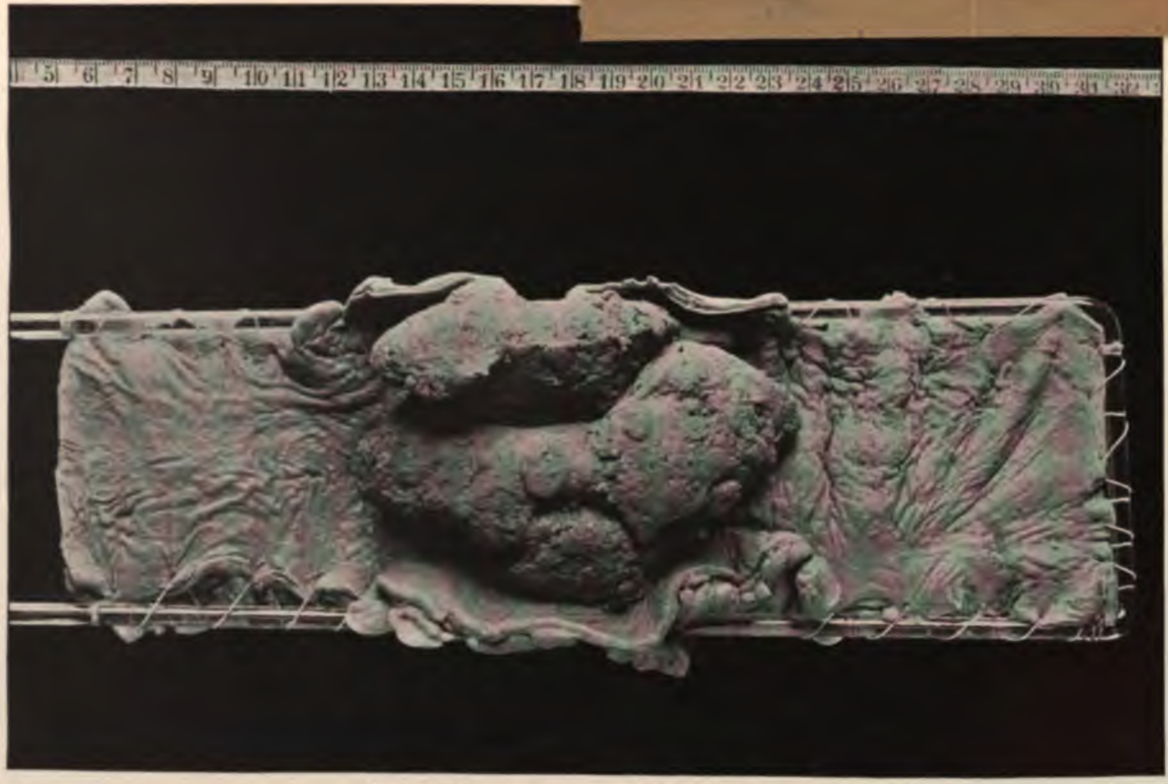


FIG. 36.—ADENOCARCINOMA OF SIGMOID FLEXURE.

per cent. of intestinal cancers occur in the rectum; 15 per cent. he large intestine; and 5 per cent. in the small intestine.

I do not consider it a part of the object of this work to give a tical consideration of the various theories concerning the nature malignant neoplasms, particularly of the polemic, that has re-ntly been reopened by Ribbert—whether the carcinoma is of epi-ielial or connective-tissue origin. Concerning these subjects I fer to the corresponding literature, which can be found in the rticles of R. Beneke ("Schmidt's Jahrb.," Bd. CCXXXIV, 1892, S. 3 u. 185), Stick ("Berlin. klin. Wochenschr.," Bd. X, No. 37 u. 38, 1873), Hauser ("Das Cylinderepitheliom des Magens u. Dickdarms," Jena, 1890), and von Nothaft ("Arch. f. klin. Med.," Bd. LIV, S. 555). The better known theories attempting explanation of malignant neoplasms have been compiled in a recent work by Hansemann ("Die Bösartigen Geschwülste") and in Ribbert's "Lehrbuch d. allg. Pathol.," S. 532. The principal hypotheses are the following: that of Tiersch, who advanced the view that the histogenetic equilibrium of the tissues becomes disturbed with advanced age, and thereby the weakened connective tissue is overcome in the struggle with the more resistant epithelial tissue that has been excited to proliferation by trauma. Rindfleisch suggested that the abnormal tendency to growth in the malignant epithelial cell, although originally provoked by trauma or chronic inflammatory processes, was due to the loss of the regulatory influence of the nervous system upon the rate of growth. Klebs advanced a theory in his work on "Pathology," according to which the proliferation of the cancer cell was caused by a kind of cell fertilization, brought about by penetration of leukocytes into the nuclei of normal cells, there becoming dissolved, and acting as the morbid stimulus to growth. According to Cohnheim, carcinoma arises from disrupted and disseminated embryological germ cells, which lie dormant in the various tissues until they are incited to growth by various irritants (trauma, etc.). The ingenious hypothesis of Cohnheim, which has received the most support among all the various explanations, and according to which the origin of the cancer is to be explained by a proliferation of embryonic germinal cells, can not be experimentally proved, and if we accept it, it is hard to understand why those germinal cells usually begin to proliferate only in advanced age, and cancer is not more frequently observed in the case of those persons who are repeatedly subject to acute and chronic catarrh of the intestines. Sometimes the cancer, as has been remarked previously, develops on the basis of an ulcer of the duodenum, an observation which seems to argue rather against than for the theory of Cohnheim, since the assumption seems justified that those embryonic germinal cells left behind have been completely destroyed by the ulcerative process, especially if the same has extended to the serosa and the cancer starts from the scar. The view of Virchow ("Die krankhaften Geschwülste") that carcinoma is due to the local disposition of growth incited by trauma has received the largest number of adherents. In the tissues there are certain regulatory processes which control the normal growth of cells. The local disposition which Virchow speaks of is an arrest of this normal regulatory control of cell growth. It may be congenital, or acquired by various disease processes, inflammations or ulcerations, or by trauma. According to Billroth, Beneke, Frerichs ("Virchow's Archiv," Bd. XXXVI, S. 86), Tiersch, and Luecke, a general specific constitutional disposition is necessary if a trauma is to provoke a carcinomatous neoplasm. M. Gockel ("Archiv f. Verdauungs-Krankheiten," Bd. II, S. 461) has given us a valuable contribution concerning the traumatic origin of carcinoma, especially of intestinal carcinoma. Hitherto the profession has not attributed sufficient importance to the agency of trauma in the etiology of carcinomata of the digestive tract. With reference to carcinomata of the skin, this factor has been sufficiently appreciated. The explanation of its neglect as an etiological factor in gastric and intestinal carcinoma is, in my opinion, exclusively due to the neglect of inquiring concerning this point in establishing the anamnesis. If more attention were paid to the previous history of carcinoma patients, we should, in my opinion, have a larger number of cases in which the new growth was directly traceable to a trauma. Gockel (*l. c.*) has collected 600 cases of carcinomata from the literature in which the traumatic origin could be traced beyond objection. In 60 cases of carcinoma affecting the intestinal tract he found that 7 (11.6 per cent.) presented such a history. A similar report is made by Boas ("Verhandl. d. XVIII. Cong. f. innere Med.," Wiesbaden, April, 1900, S. 300–374); he reports a personal experience with 200 cases of undoubted carcinoma, in 18 of which he was able to

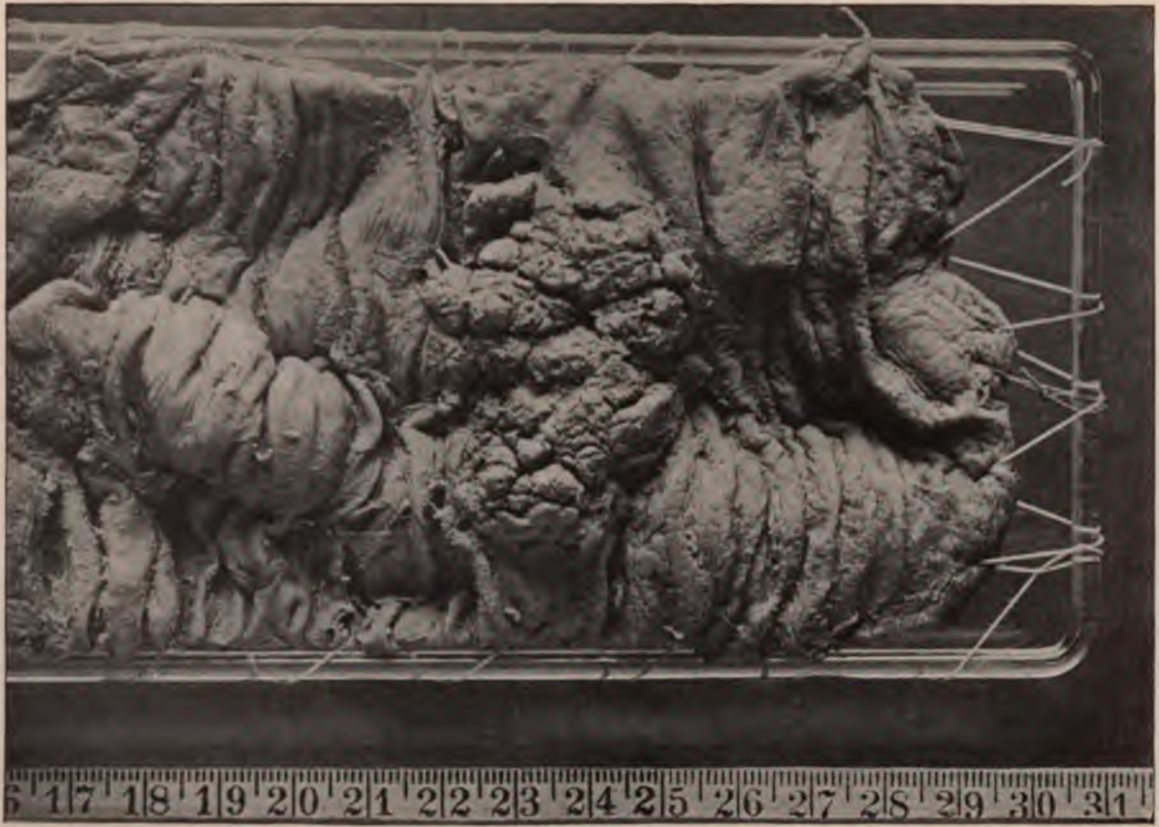


FIG. 37.—ADENOCARCINOMA OF COLON WITH PSEUDOMEMBRANOUS COLITIS.

trace the occurrence of malignant neoplasms in the direct ascendency, and to fix the factor of trauma in 19 cases as one deserving a place in the etiology. In my own private and hospital practice the element of trauma was inquired into and reported in 112 cases of gastro-intestinal carcinoma. In 15 of these a positive history of trauma as of etiological moment had been recorded. Carcinomata which develop several days or several weeks after a trauma should not be attributed to this factor, but those that develop after a period of time varying from one month to a year after the experienced trauma should be logically brought into causative relation therewith. In three of my cases of intestinal carcinoma the first symptoms of the neoplasm were not experienced until one year, fourteen months, and two years respectively had elapsed.

It is necessary to enlarge our conception and definition of trauma in its application as an etiological factor in the causation of neoplasms. In general we may define trauma as an influence upon the organism by force or power, independently of kind, locality, strength, or duration. A weak application of force, if it exists a long time, may exert as deleterious an influence as a strong application exerts for a short time. The trauma, therefore, may be acute or chronic, direct or indirect. It may act from the exterior or from the interior of the organism. Foreign bodies swallowed with the ingesta may exert the same effect upon the stomach as a blow from the exterior. A continued irritation by excessive hyperchlorhydria may exert a similar deleterious influence as constant pressure against the stomach from the outside. Among the more common traumata are to be mentioned pressure, blows, pushes, contusions, falls, bites, mashes, stabs, cuts, burns, caustics; these are acute direct traumata. Then follow the more slowly acting traumata, such as carrying of heavy weights, strong retching, vomiting, powerful action of the abdominal muscles, biliary, renal, and fecal concretions, the irritations of the secretions or excretions—anything that rubs, scrapes, scratches, or irritates the tissues, either physically, chemically, or thermically. None of the theories which we have mentioned is proved, but I wish to emphasize the agency of trauma in the causation of intestinal carcinomata. Nothnagel (*l. c.*, p. 219) calls attention to the fact that intestinal carcinomata, similar to gastric carcinomata, may arise from ulcers. He had in a patient diagnosed a dysenteric cicatrix in the splenic flexure of the colon. Billroth operated for stenosis caused by this condition. The cicatricial stricture was found, but close by it was a carcinoma which had been formed in the cicatrix.

Age.

Carcinomata are most frequent from the fortieth to the sixty-fifth year. It is very well known, however, that they may occur before the fortieth year; thus, Nothnagel observed a case of carcinoma of the cecum in a boy twelve years old; Schōning two cases of rectal carcinoma in girls seventeen years old; and Clar reported a case of medullary carcinoma of the colon in a boy three years old. Maydl ("Ueber den Darmkrebs," Wien, 1883) has drawn valuable conclusions from the cancer material of the Allgemeines Krankenhaus in Vienna. One-sixth of all intestinal cancers occur between the ages of thirty and forty, and one-seventh of all intestinal cancers occur before the age of thirty. These figures are sufficient to show that it is not safe to exclude intestinal cancer in a concrete case simply because the patient is young.

Types and Structural Effects of Intestinal Malignant Neoplasms.

In by far the great majority of cases intestinal carcinomata are primary. There are very few authentic cases of secondary metastatic carcinomata. The most frequent form, according to Hauser (*l. c.*), is the cylindrical epithelial carcinoma. The next most frequent form is the medullary carcinoma, which most often gives rise to disintegration and ulceration of the neoplasm. The third rarest form of malignant neoplasm is the colloid carcinoma, and the rarest of all is the scirrhus. The histological structures of these various types of malignant neoplasms have been described in the author's work on "Diseases of the Stomach," second edition, pp. 527–546. It is rare that intestinal carcinomata form isolated nodules or tumors of any magnitude. They rarely develop into extensive or diffuse infiltrations. Their most characteristic form is that of a circular or girdle-shaped ulcerating neoplasm. The soft medullary cancer is the type which most frequently makes tumors of considerable size, while the scirrhus is characterized by infiltration of the walls of the intestinal canal. Cancers of the bowel exhibit a tendency toward ulceration, and consequently to the causation of hemorrhage and suppurative disintegration. In rare cases sequestration of portions of the neoplasm has been observed. As a consequence of the frequent circular shape of the neoplasm, stenoses of the bowel are frequent. Such stenoses may be absolute, and be followed by all the symptoms of suprastenotic dilation and hypertrophy of the muscularis that has been described in the section on Enterostenosis and Occlusion. The efforts of the powerful muscular contractions as a result of the muscular hypertrophy may temporarily effect a permeability of the stenosis. The course of the apparent intestinal occlusion is then one of alternating improvement and aggravation. Eventually the contractile power of the compensating hypertrophy is exhausted, and the clinician is confronted with a pronounced case of paralytic occlusion. In rare instances the overwhelming efforts of the suprastenotic muscular hypertrophy have caused a rupture in the weakest portion of the diseased intestine.

Location.

The statistics of Bryant ("Annals of Surgery," February, 1893) are based on 110 autopsies. Of these, the carcinomata were found in the small intestine 6 times, in the cecum and ileocecal region 7 times, in the transverse colon and hepatic and splenic flexures 19 times, and in the sigmoid and rectum 78 times.

The following statistics I have compiled from the reports of Maydl ("Ueber dem Darmkrebs," Wien, 1883), taken from the autopsy reports of the Pathological Institute of the Wiener Allgemeines Krankenhaus from 1870 to 1881, in which time there were 20,480 autopsies, with 1406 cases of carcinoma in general, of which 100 were found in the intestines. Next the reports of the same institute, collected by Nothnagel and compiled from 21,358 autopsies, in which there were 2125 cases of carcinoma in general; of these 243 were found in the intestines. In twenty-four years, therefore, 343 intestinal cancers came to autopsy in this institute; 7 were in the duodenum, 10 in the ileum, none in the jejunum, 164 in the colon, and 162 in the rectum. Müller's statistics are taken from the Pathological Institute of Bern, 1886 to 1891 ("Beitr. z. Kenntniss d. Metastasen-Bildung malign. Tumoren," Inaug. Diss., Bern, 92), during which time there were 5621 autopsies, of which 521 were carcinomata in general.

Georg Heimann ("Arch. f. klin. Chir.," 1899, Bd. XLVII) has recently collected the statistics of carcinomata of the digestive organs from all the general hospitals of Prussia in 1895 and 1896. There were in all 20,544 cancer cases; of these 10,573 had attacked the intestinal canal. The figures of this statistician are given in the following table.

The statistics quoted from A. Zemann were published in "Bibliothek. med. Wiss.," by Drasche, Bd. III, H. 1 u. 2, S. 49. They are based upon 21,624 sections in the Wiener allgemeines Krankenhaus. The cause of death was neoplasm in 2070 cases. Of these 1744, or 80 per cent. of all neoplasms, were carcinomata. Of the 1744 carcinomata 912—that is, 52 per cent.—were found in the intestinal tract, beginning with the tongue and extending to the anus.

COMPILATION OF STATISTICS OF LOCATION OF CARCINOMATA IN THE DIGESTIVE TRACT.

	A. Zemann.	Leichtenstern.	Maydl.	Bryant (Intestinal Carcinomata Only).	Müller.	Reports of Pathological Institute in Vienna, 1882–93, Collected by Nothnagel.	Geo. Heimann, Autopsies on General Carcinoma Cases Only.
Number of autopsies,	21,624		20,480	110	5621	21,358	20,544
Tongue,	37						269
Pharynx,	34						
Mouth and pharynx,							192
Esophagus,	136						1011
Stomach,	540						4288
Duodenum,	3	17	2	6	9	5	
Jejunum,							
Ileum,	6		4			6	
Middle ileum,		3					
Lower ileum,		13					
Ileocecal valve,		9		7			
Cecum,	12	20	9			14	
Ascending colon,		6	6				
Transverse colon and flexures,		30		19			
Descending colon,		11					
Colon,	32		17		13		
Vermiform process,	1	3	1			1	
Sigmoid flexure,	30	42	13	78		40	
Rectum,	81	616	48		19	114	1204
Colon in general,						63	
Intestine in general,							1706
Liver and gall-bladder,							979
Pancreas,							92

From the foregoing chart and description it will be observed that of the total number of general autopsies as collected by A. Zemann, Maydl, Müller, and Nothnagel, amounting in the aggregate to 69,083, there were 5796 deaths from carcinoma—about 8.4 per cent.

Of these 5796 cases of carcinoma, 1296 occurred in the intestinal tract. Thus it will be observed that 22.35 per cent. of all carcinomata occur in the intestinal tract.

Thirty-five of these intestinal carcinomata (2.7 per cent.) occurred in the duodenum, jejunum, and ileum; 35 (2.7 per cent.) were in the cecum; 131 (10.11 per cent.) in different parts of the colon; 3 (0.23 per cent.) in the vermiform process; 83 (6.405 per cent.) in the sigmoid flexure; 262 (20.22 per cent.) in the rectum. Thus it appears that carcinomata are found in the rectum oftener than in any other part of the intestinal tract.

However, according to the statistics of G. Heimann, as given in the above table, 51.5 per cent. of all cancers occur in the intestinal tract, and of these about 11.39 per cent. attack the rectum.

Metastases.

Intestinal carcinomata give rise to the formation of similar neoplasms in other tissues and organs by dissemination through the blood- and lymph-vessels. In cancers of the rectum, the most frequent locality, the metastases occur first in the retroperitoneal, the sacral, and lumbar lymphatic glands. The inguinal lymph-glands show cancerous involvement only when the original carcinoma reaches very far down in the rectum or has involved the skin of the anus. Orth ("Spec. path. Anatomie," Bd. 1, S. 852) asserts that the intestinal cancers extend relatively early to the lymph-glands and peritoneum, and cause metastases in the liver and other organs at an early period after their original development. Hausmann (*l. c.*), Maydl (*l. c.*), and Rüpp, however, argue that intestinal cancers give rise to metastases relatively late in their development. This, they claim, is particularly so with regard to the rectal carcinoma. Kraske found no metastases in 6 out of 12 cases of rectal carcinoma, and Iversen (*l. c.*), who investigated 47 rectal carcinomata at autopsy, found the glandular organs free of metastases in 21 of these. Personally I have records of 8 autopsies on rectal carcinomata, in 4 of which no metastases could be found in the lymph-glands or other organs. As this matter of the early or late development of metastases is of great interest from a surgical standpoint, it is of importance that more attention should be paid to this point hereafter at autopsies and operations and the results reported.

The various kinds of intestinal carcinomata which have been described in the preceding do not give rise to metastases with the same frequency; nor do they all form metastases in the same organs with the same frequency. For instance, the large medullary carcinomata, according to Hauser (*l. c.*), form metastases principally in the regional lymph-glands, and the scirrhus, which may be of very small size, gives rise to extensive metastases in the liver. Lastly, the gelatinous or colloid carcinoma rarely causes metastases in internal organs, but frequently forms them in the bones, lymph-glands, and peritoneum. Orth, however, pictures a case (*l. c.*, p. 852) of colloid carcinoma of the rectum in which there were numerous metastases in the liver, lungs, bones, and adrenal bodies. Concerning this colloid or gelatinous carcinoma, it might be stated here that as its surface and stroma are soft, similar to granulation tissue, it frequently experiences considerable losses of substance; as the rigidly infiltrated wall of the rectum can not collapse, extensive rectal dilations are formed thereby. The characteristic mucoid masses of this cancer which result from its ulceration may be discovered in the stools during life. According to Charon and Legedank ("Jour. de Med. de Bruxelles," 1877, p. 514), they can be recognized by their form and structure, for the mucoid lumps are arranged in spherical alveolar masses in which concentric granular striations are recognizable.

When intestinal cancers ulcerate into blood-vessels, infectious protoplasmic masses are transported to the circulation. As the roots of the portal vein are located directly in the intestine, and therefore may be in communication with the neoplasm, this explains why we meet more often with metastases in the liver than we do in the kidneys, spleen, lungs, uterus, and ovaries. Metastatic carcinomata of the mesenteric or omental lymph-glands may cause secondary intestinal occlusions, as well as disseminated carcinosis of the peritoneum.

Adhesions and Agglutinations.

These may give rise to complications resembling the clinical picture of intestinal occlusion, or they may prevent septic peritonitis by avoiding perforation. Fistulous communication between the intestine and the colon, bladder, and genital organs have been described. In rare cases perforations communicating with the external skin may occur. Similarly one intestinal loop may communicate with another by this process. These peritonitic adhesions and agglutinations sometimes cause great operative difficulties, for in my experience it is impossible to diagnose them in the majority of cases, particularly when a number of intestinal loops, together with their mesenteries, have become adherent. Remarkable distortions of the viscera can be created by these processes. I have observed a case of carcinoma of the sigmoid flexure in which the sigmoid, rectum, right ovary, uterus, loops of the small intestine, peritoneum, and omentum were agglutinated in one large conglomeration. Nothnagel (*l. c.*) describes agglutinative conglomeration of the hepatic flexure of the colon in which the primary neoplasm was seated, with the peritoneum, omentum, small intestine, and cecum; another instance in which the carcinomatous cecum was adherent to the sigmoid flexure. The cecum may be drawn over to the left side, and the sigmoid flexure, to the right side.

In 280 cases of intestinal cancer collected by Courtois-Suffit ("Traité de Médecine," by Bouchard and Brissaud, tome IV, p. 505), 28 cases occurred in the small intestine. For every case of cancer of the small intestine one observes 9 or 10 cases of cancer of the large intestine. The 280 cases were distributed as follows:

Jejunum,	4
Ileum,	21
Cecum,	48
Ascending colon,	22
Hepatic flexure,	8
Transverse colon,	26
Splenic flexure,	10
Descending colon,	29
Sigmoid flexure,	100

Pathologic Anatomy.

Since intestinal cancer shows in its inner construction and outer appearance the same behavior as cancer of the stomach, I may refer to the pathological anatomy of the latter (Hemmeter, "Diseases of the Stomach," second edition, pp. 527–565). I must emphasize here again that the intestinal malignant neoplasm very often is a cylindrical-celled carcinoma with glandular architecture, —adenocarcinoma,—which starts from the epithelia of the glands of Lieberkühn. Fairly often colloid cancers ("Gallert carcinom") are found in the rectum, while, on the contrary, melanotic cancers—melanocarcinomata—are found very rarely. Sometimes the cancer takes its start from the pavement epithelia of the anus,—cancer of the pavement cells (platten-zellen Carcinom) or cancroid,—and this extends preferably to the perineum and the vagina. In the proportion in which the connective tissue or the cells predominate in the neoplasm it shows a hard, sometimes cartilaginous, consistence, —scirrhus,—or it is more of a semisolid, dry, or of a moist constitution. The colloid cancers contain a brownish, colloid, and thick liquid. While the scirrhus, which has a tendency to shrink, usually grows but slowly, the cancers that are rich in cells can grow very rapidly, and in the latter a partial disintegration of the central portions often occurs under the influence of the digestive secretions, as well as in consequence of changes in the blood-vessels leading to local thrombosis and embolism and the formation of so-called cancerous ulcer. It has already been stated that intestinal cancer, if it occurs as a primary disease, usually causes a stricture of the intestine. If this has reached an advanced stage, the intestine above the stricture is excessively distended by the stagnating fecal masses and gases, its walls are considerably thickened, and the result is an advanced hypertrophy of the muscles. Catarrhal inflammations and ulcers are often found in the immediate neighborhood of the stricture, for very apparent reasons. In the case of extreme distention of the walls of the intestine above the stricture a rupture of the intestine may take place. Below the stricture the wall of the intestine becomes thinner, and the latter, as soon as no more chyme reaches it, contracts very much. If a considerable disintegration of the ulcer causing the stricture takes place, the intestine may become passable for a while (spontaneous cure of Rokitansky). However, since the connective tissue which is formed in place of the disintegrated portion has a strong tendency toward retraction, the intestine soon becomes stenosed as before, with this distinction, that the first enterostenosis was caused by the substance of the neoplasm itself—it was really an "obturation"; but the second is a stricture due to the contraction of cicatricial tissue. It is easy to understand that during a partial breaking up of intestinal cancers erosion of the blood-vessels and severe loss of blood may occur. In some cases the cancer of the intestine is not confined to the mucous or the submucous membranes, but extends to the muscular layer (concerning the secondary changes of the latter refer to article on "Cancer of the Stomach," in the author's work, *l. c.*) and to the serosa.

If this, however, is perforated before an agglutinative adhesion with neighboring parts was possible, fatal general peritonitis occurs. Since, however, the cancer usually extends but slowly to the deeper lying layers, usually there is sufficient time given for the development of adhesions, by which general peritonitis is prevented. If the intestinal cancer extends to the peritoneum, cancerous peritonitis with hemorrhagic exudation may be the result. Cancer of the intestine, in extending to neighboring organs, may produce abnormal communications after the disintegration of the neoplasm. In this way fistulous communications between the stomach and the colon, external fistula of the intestines opening upon the outside of the abdominal wall, may arise; furthermore, fistulæ between the rectum and the bladder (cystitis); fistulæ between the rectum and the vagina and the uterus may develop. Cancer of the rectum usually causes chronic proctitis and the development of hemorrhoids. In cancer of the duodenum the other resulting conditions are the same entirely as those discussed more fully in chapter XXX on Stenosis of the Duodenum. I therefore refer to the same, emphasizing here the fact once more that cancer of the duodenum may cause continuous jaundice and distention of the stomach. In many cases the cancer either extends directly to neighboring organs, or cancer metastases in the lymphatic glands, the liver, and other organs develop. Sometimes cancers are found at two different places in the intestine,—for example, in the rectum and in the cecum,—but the occurrence of two circular cancerous strictures at different parts of the intestine has probably never been observed.

Symptomatology.

Introductory Remarks.—The disease always develops so slowly and chronically that its commencement in all cases eludes proof. If the intestinal cancer is situated at a place difficult of approach, and if it causes but slight disturbances on the part of the intestine, a fact often observed in people of advanced age,—for instance, if only constipation occurs, from which the patient suffered in previous years,—then the disease may remain completely latent during life and be discovered only at the autopsy. Often the suspicion is awakened that a malignant neoplasm exists on account of increasing cachexia and anemia, in spite of an undisturbed appetite and sufficient nourishment. In other cases, to be sure, annoying subjective complaints—unpleasant sensations of pressure and tension in the abdomen, colic-like pains, or a circumscribed sensibility to pressure, as well as abnormal suppression of the evacuations—manifest themselves. Frequent attacks of constipation, which occasionally alternate with diarrhea, when occurring in conjunction with these symptoms indicate an affection of the intestine, although the symptoms do not give certain proof of the malignant nature of the same. Suddenly the symptoms of an intestinal obstruction appear, with or without any apparent cause. The autopsy shows a cancerous stricture of the intestine. There are cases of peritonitis with hemorrhagic exudation, and only at the autopsy is the fact discovered that this was secondarily caused by an intestinal cancer.

The development and progress of the diseases in general are about as follows: The first symptoms, which may appear in a shorter or longer time after the commencement of the cancer, consist of subjective complaints which express themselves in unpleasant sensations of oppression, fullness, and tension in the abdomen. After some time the patients complain of periodic colicky pains or a circumscribed sensibility of pressure, and spontaneous pains at the same place are occasionally described. Sometimes the patients are annoyed only by a dull pressure in the lower part of the abdomen. The appetite usually remains undisturbed for a considerable period of time, being sustained longest when the neoplasm is near the rectum; only in those cases where the cancer is in the duodenum do early complaints about the functions of the stomach, anorexia, nausea, vomiting, cardialgic pains, and finally the signs of a distention of the stomach, manifest themselves. In spite of sufficient nourishment the patients become anemic and emaciated, with edema at the joints or knuckles, feel tired and depressed, and indisposed for mental or bodily exertion. Of course, these symptoms, even if they continue a long time, do not suffice to justify a certain diagnosis of intestinal cancer, especially since the emaciation may be entirely lacking or very slight in a rapid course of the disease. The assumption of an intestinal cancer is justified only when unmistakable signs of a stricture of the intestines can be demonstrated, or when a hard, nodular tumor that is sensitive to pressure can be felt and tumors of other organs can be excluded. Malignant tumors of the duodenum vary in their symptomatology according to their location—that is, according to their situation above or below the papilla of Vater. The signs and symptoms will then be those of either infra- or suprapapillary stenoses of the duodenum (see chapter on Enterostenoses). When the neoplasms are at or about the orifice of the pancreatic or common gall-duct, they naturally lead to eventual occlusion of these channels, and, in the case of the gall-duct, to jaundice; not invariably so, however, for A. Descos and L. Béviel ("Revue de Méd.," August 10, 1899) have reported a singular case of duodenal carcinoma involving the biliary passages and the head of the pancreas without having caused progressive icterus.

Intestinal Stricture.—If the hindrance which the stricture of the intestine offers to the passage of the feces and the chyme downward is not yet very great, it may be for some time overcome by energetic contraction of the hypertrophic muscles of the section of the intestine lying above the stricture. To be sure, the evacuations occur less regularly and are of a firmer consistency than before; sometimes it is necessary to administer an aperient, but a longer retention of the feces is prevented especially if the nourishment is judicious and adapted to the case. On the other hand, even if the stricture is only partial, if the diet is faulty,—for instance, after eating a great amount of leguminous vegetables,—when the undigested portions and the feces accumulate above the stricture, as soon as the intestine is completely impassable the symptoms of intestinal obstruction appear, to which the patient may succumb. It may also come to a lesion of continuity of the intestine, either in the neighborhood of the stricture or also in distant parts of the intestine, especially if the bowel is tympanitically distended by gases.

If the mucous membrane of the intestine above the stricture is strongly irritated by the stagnating feces as well as by gases, diarrhea may set in, which may periodically alternate with constipation. Periods of diarrhea and constipation may recur, each lasting for some time. In one case of cancer of the cecum related by von Leube, diarrhea lasting for months was observed. Similar cases of carcinoma of the descending colon and sigmoid have been described by Nothnagel and the author (see chapter on Enterostenosis).

Condition of the Evacuations of the Bowels in Intestinal Stricture.—When the intestine is subject to considerable stricture, the feces often show a flat, compressed, stringy, or spirally wound form, because only small amounts of excrement can pass the stricture at one time. Small globules, like the excrement of sheep, may be evacuated, which probably are formed in the haustra of the large intestine. The aforesaid form of the feces is, however, not characteristic of an intestinal stricture (see article on Constipation and on Feces, in chapter on Occlusion or Enterostenosis), since it might also occur in spasmodic contractions of single loops of the intestines, as well as during long-continued inanition, when the empty intestine contracts very much. These abnormal shaped feces are of importance for the diagnosis only if normally formed masses of fecal matter are never evacuated in the intervening time. After a long stagnation of the feces in the intestines the excrement often gives off a very offensive odor, and the flatus is also very putrefactive. The excrement becomes positively as offensive as carrion if a disintegration and decay, suppuration, and ichorous degeneration of a part of the cancer have occurred. Furthermore, the evacuations often contain greater or less amounts of mucus, pus, and blood. The admixture of the mucus is not a result of the cancer of the intestine itself, but of the catarrh occurring in the vicinity of the neoplastic stricture. If the cancer is in the first section of the large intestine or in the small intestine, the mucus is slightly mixed with the excrement; whereas if the cancer is located in the lower section of the intestine and in the rectum, the mucus is usually distributed on the surface of the fecal matter. Pus is found in the evacuations in the stage of partial disintegration and suppuration of the intestinal cancer, as well as in extensive ulcers and severe inflammation of the mucous membrane, especially that of the rectum. The absence of pus is not an absolute proof against the existence of cancer or ulcer, because in long stagnation of the feces in the intestine the leukocytes (white blood-corpuscles) can disintegrate and may be reabsorbed, or the amount of pus formed may be so small as to escape detection.

Blood is found in the evacuations in erosions of blood-vessels as well as of ulcers and hemorrhoids. If it takes its origin from the upper section of the intestine, it often escapes detection even in the microscopic investigation if the amount is not great, because it is then mixed with the feces and is changed by decay. In this case it can be detected only chemically—by the guaiacum test—or by a spectroscopic examination. Small portions of cancerous tissue have repeatedly been found in the feces, as well as in the liquid used for lavage of the colon. If these have the size of a bean, of a cherry, or even of a walnut, as has occasionally been observed, they can hardly escape detection in a careful examination of the feces, while, on the contrary, small particles of the cancerous tissue might often be overlooked. In doubtful cases the occurrence of parts of cancerous tissue in the evacuations is naturally of supreme importance for the recognition of the neoplasm. In all suspected cases I urgently recommend washing the stools in the Boas stool-sieve, as described by Dr. Harry Adler in the chapter on Examination of the Feces.

If the intestinal stricture has reached an advanced stage, complete suppression of the evacuations finally occurs. If the cancer is in the lower part of the intestine, the suppression may be endured for a long time. In two cases cited by Cooper and Hensgen complete suppression of the evacuations existed for forty-four and eighty-eight days respectively. These cases appear strange at first sight. Every physician who has once had the opportunity to notice the enormous expansion of the entire colon in cancerous stricture of the descending colon can well understand that it can easily hold very considerable amounts of chyme and feces, especially since the same is very much thickened by the reabsorption of water and decreases in volume. The evacuations also cease completely in cancer of the duodenum as soon as the stricture has reached an advanced stage, because no chyme reaches the lower parts of the intestine thereafter.

In such cases the region of the stomach is usually very much distended, and the remainder of the abdomen is retracted, because the empty intestines contract. If the cancer of the duodenum is in the neighborhood of the opening of the common gall-duct, continuous jaundice sets in, and the evacuations, as long as they still occur, become achylic. If, in cancer of the duodenum, jaundice does not set in, the evacuations may be perfectly normal, or sometimes they contain slight amounts of mucus.

Cases are reported in which, after persistent suppression of the evacuations as a result of cancer of the small or large intestine, suddenly for a short time large amounts of evil-smelling excrement were passed off which contained mucus, pus, and often blood. This has occurred in two cases at my clinic, and it indicates that the stricture has become passable for a short time again as a result of a partial disintegration of the cancer, or that abnormal communications between the section of the intestine in question and lower portions of the intestines have developed.

Occurrence of a Tumor.—Frequent and methodical palpation of the abdomen in cases of suspected carcinoma is of extreme practical importance. When the abdominal walls are too rigid, the palpation should be carried out while the patient is in a warm bath or under narcosis. If this examination does not lead to the desired end, a combined palpation of the vagina and the rectum is indicated in the case of women ; in the case of men, examination of the rectum alone is to be made, since it is unconditionally required to prove or exclude the existence of a cancer in that location. The more trained the hands of the physician are in palpation, the more rarely the intestinal cancer will escape discovery. The tumor formed by the cancer of the intestines has the following characteristics, which must be considered during the examination: At first smooth, round, or oval, it becomes uneven, lumpy, and also sensitive to pressure after it has existed some time. In smaller tumors one feels only an indistinct resistance located in the depth of the abdomen. The tumor seldom becomes so large that it causes the walls of the abdomen to project at any one place, those cases in which it does this being usually cancers of the cecum. If the tumor is situated on larger abdominal vessels, the arterial pulsation is perceptible through it. If it is situated on sections of the intestine that are freely movable,—for instance, on the horizontal portion of the duodenum, on the jejunum and the ileum, or on the transverse colon and its flexures,—unless it has grown together with neighboring organs it usually is capable of being moved and shoved about. Since those sections of the intestines just named become heavier when the cancer has attained considerable size, they sink downward, and consequently tumors of the small intestine and of the transverse colon are usually found in the hypogastric region downward from the navel. Cancers in the lower part of the duodenum and of the cecum, as well as cancers of the rectum, are not at all movable, or movable only to a very slight degree. Osler calls attention to the hardening during contraction of the hypertrophied wall in the vicinity of the growth, meaning the compensatory hypertrophy in the suprastenotic loops, and the bubbling of gas which may be heard as well as felt ("Diagnosis of Abdominal Tumors," p. 117). Case 44, which he describes in detail (p. 111), presented this latter sign to a degree in which it is rarely observed. It can occur only in stenosis, not in absolute occlusion, and as the autopsy, by Flexner, revealed in Osler's case, the hepatic flexure of the colon in which the tumor was found had not been narrowed to any considerable extent. Although the primary cancers of the intestines, which usually are circular, directly cause strictures, larger cancerous masses—that is, particularly in secondary cancer—may also exert pressure on a neighboring intestinal loop and cause compression of the same. Of course, tumors of other abdominal organs—the mesenteric glands, omentum, and kidneys—may also cause this same effect. The tumor of intestinal cancer usually, as a result of thickening of the walls of the intestines, of the greater massing of feces and gas above the neoplasm, and of a growing together with neighboring organs, appears much larger than it actually is. Therefore its size varies considerably in the same patient at different times, according to the amount of fecal matter that has accumulated in front of the growth. On account of the great movability of intestinal neoplasms, except in the localities referred to, and also because loops of the intestine filled with air and gas often push themselves between them and the abdominal wall, which becomes more or less distended, intestinal tumors seem larger at one time and smaller at another. At one time a tumor is felt more plainly; at others less plainly or not at all. Sometimes it is not perceptible for several weeks at a time, so that the physician may already have hope that he made a wrong diagnosis until it again becomes perceptible by palpation, often enlarged. For the reasons just stated percussion does not give very reliable results. In some cases the tumors give forth a muffled sound when the finger or the pleximeter is impressed deeply upon them during the percussion. The conservatism and caution necessary before making a positive diagnosis of intestinal neoplasm are nowhere better illustrated than in the article on "Abdominal Tumors" by Osler (*l. c.*). Case 44 above cited seems to have been under observation from August 3 to October 20, 1892, and on October 12th there was still a discussion as to whether it was a case of renal, hepatic, or intestinal tumor (*l. c.*, p. 111).

In contrast to fecal tumors the tumors conditioned by the intestinal cancer do not yield to pressure of the finger and do not change their form. Besides, in the fecal tumors one often observes a slow advance downward. Sometimes several of them exist at the same time, whereas in intestinal cancer it is extremely rare to find two growths at the same time (see chapter on Obturation by Fecal Concretions). After the successful use of aperients or enemata the fecal tumors become smaller or suddenly disappear. Sometimes cases occur where the diagnosis between cancer of the intestines and fecal tumor is exceedingly difficult, because the latter may also feel hard and lumpy, remain at one place for a long time, become sensitive to pressure, and not decrease at all or but very little even after a considerable evacuation, because only the central parts of the feces are softened and evacuated, a hard peripheral shell remaining attached to the intestinal wall. In such cases a differentiation of the two diseases is possible only after a long period of observation. The fecal tumor, however, finally decreases and entirely disappears after repeated administration of aperients and frequent irrigations.

Cancer of the Rectum.—This may usually be diagnosed earlier than cancer of other parts of the intestines, because its more striking symptoms and the following conditions are extraordinarily characteristic: Violent tenesmus; pains during evacuation; alternating evacuation of excrement and of pure mucus, pus, or blood; violent pains radiating toward the loins, the genitals, and the upper parts of the thigh; the occurrence of hemorrhoids. To be sure the above-mentioned symptoms in many cases appear only a long time after the beginning of the cancer of the rectum, especially if it ulcerates somewhat late. The diagnosis is rendered much easier by a digital investigation of the anus and the use of the rectal speculum (see section on Diseases of the Rectum, by Thomas Charles Martin). Since hemorrhoids hardly ever fail to appear in cases of cancer of the rectum, the physician must not omit a digital examination of the rectum if these hemorrhoids occur and at the same time some of the other symptoms appear. If this examination, as well as endoscopy, gives a negative result and cancer of the rectum is still suspected, then the examination must be made under chloroform or ether narcosis. This, however, just as the digital examination, must always be made with great precaution, since the intestine is occasionally so thinned and brittle that it is easily ruptured when strongly irritated by mechanical means. If the rectal cancer exists a short time only, small smooth knots are felt during the digital examination, over which the mucous membrane can be but slightly moved. If pressure is exerted on the abdomen during the examination, the knots are frequently felt more distinctly. After a longer existence of the cancer of the rectum the knots increase in size and the formation of a cancerous ulcer, the surface of which feels rough and fissured, occurs. These so-called carcinomatous ulcers of the rectum are due to disintegration of the central or peripheral portions of the neoplasm. Later on the rectum is gradually transformed into a hard, funnel-shaped, stiff-walled cylinder, narrowing toward the top, or a roundish, conical tumor projecting downward is felt, which bears a great resemblance to the vaginal portion of the uterus. The finger introduced is often soiled with an offensive mass of mucus, blood, and pus. With the further development of the cancer of the rectum the subjective complaints of the patient often increase to such a degree that they render him desperate. The radiating neuralgic pains become exceedingly violent, tenesmus occurs at shorter and shorter intervals, forcing out under strong pressure pure mucus or mucus mixed with pus or blood. If the cancer is situated very far down in the rectum, a prolapse of the tumor, together with a portion of the mucous membrane, may occur during the evacuations, so that it becomes directly visible to the eye. If the inflammation spreads to the integument of the sphincters, the patient frequently complains about painful cramps of the sphincter. Eventually paralysis of the sphincter sets in, and a thin offensive liquid mixed with blood and pus passes forth from the anus continually, attacking the external skin and producing dermatitis. Since the pain in cancer of the rectum becomes extreme during evacuations, the patients often suppress the desire for evacuation arbitrarily, and then the inflammation of the rectal mucous membrane is increased on account of the greater massing of the feces; the hemorrhoidal formations are made larger, and the pain is much more intense in later evacuations, because of the condition of the feces. In many cases of cancer of the rectum persistent diarrhea exists, while in other cases the patients have rather a tendency toward obstipation. Sometimes, although anatomical changes have not yet taken place in the bladder, dysuria and also paralysis of the bladder muscles are observed. If the cancer spreads to the urinary bladder and breaks through into it (fistula of the rectum and the bladder), the irritation of the mucous membrane of the bladder produced by the feces and the intestinal gases causes an extremely violent hemorrhagic and suppurating cystitis. Feces and gases from the intestines pass forth from the urethra together with the urine, and urine also passes over into the rectum. The pain during urination is often indescribably intense.

When the rectal cancer breaks through into the vagina, feces are in the same manner passed forth through this outlet and cause purulent inflammation of the vagina and epidermis. In the same manner the cancer can continue to the uterus. If, in extremely rare cases, it breaks through into the hip-joint, acute suppurating synovitis ensues. If one of the two ureters is compressed and closed by the cancer, hydronephrosis sets in; if both are closed, uremia.

Cancer of the Duodenum.—If the tumor is located in the horizontal branch of the duodenum, it produces about the same subjective and objective signs as cancer of the pylorus, from which it can hardly be distinguished with certainty. Frequently the duodenal neoplasm, if palpated at all, can be easily moved, and gastric disturbances often appear very early—anorexia, nausea, cardialgic pains, vomiting, and, finally, the signs of gastric dilation. The contents of the stomach, as a rule, show the presence of HCl in duodenal neoplasms, but it is absent, generally, in cancer of the pylorus. If the cancer is located in the lower part of the duodenum, it is not movable at all, or only to a slight degree, and often leads to jaundice, which, being very acute, may occasionally set in with fever, vomiting, stupor, and end in death (see section on Supra- and Infrapapillary Stenosis of the Duodenum).

If the intestinal cancer is situated in the *cecum*, it usually proceeds under the clinical picture of typhlitis or perityphlitis, and one can decide only after long observation if it is a case of exudation or of a gradually growing tumor. According to von Leube, chronic exudations should decrease under the effect of poultices, while, on the other hand, cancers under the same treatment should increase.

Fever.—In simple cancers fever usually is entirely wanting. Occasionally greater or smaller elevations in temperature occur when extended inflammation, suppurations, and ulcers are secondarily caused by the intestinal cancer, or if the products of the disintegration reach the blood. Fever is, of course, observed when fecal abscesses are formed after the perforation of an intestinal cancer into the retroperitoneal connective tissue, and cause pyemia and septicemia. Finally, a secondary cancerous peritonitis, hemorrhagic cystitis, and endometritis may also be the cause of fever.

Resulting Conditions of Intestinal Cancer.—These have already been considered in the pathological anatomy of carcinoma, as well as in the chapter on Enterostenosis and Occlusion, and we will repeat only some of the most important and interesting. In intestinal cancer, as was remarked before, perforation peritonitis with closely following death or rupture of the intestine seldom occurs. A secondary cancerous peritonitis is met with somewhat more frequently (peritoneal carcinosis). Although at first this is hardly to be distinguished from a nonmalignant peritonitis, developing later on, after a longer duration of the disease, nodules of varying size can be felt through the thin abdominal wall. Sometimes the nodules on the parietal and visceral faces of the peritoneum give a grating sensation under pressure. The frictional sounds heard when the abdominal wall is moved also point to this disease. The exudation in this case is usually hemorrhagic, and upon aspiration and microscopic examination often shows characteristic cells presenting evidences of atypical or pathological mitoses.

Fistula of the Stomach and the Colon.—Very characteristic symptoms are caused when a broad communication has been established between the stomach and the transverse colon after a firm adhesion of both organs. If both the passage from the stomach to the large intestine and, vice versâ, the passage from the colon to the stomach are free and no valve-like impediment exists, food and drink, immediately after their introduction into the stomach, pass over into the large intestine, usually causing diarrhea, and the diarrheic evacuations contain large amounts of food remnants not at all or but very slightly digested (lienterie). Feculent vomiting often occurs. When water is poured into the stomach through the tube, it is not collected at the bottom of the stomach, but quickly passes off by the rectum. The inflation of the stomach by means of air or carbonic acid gas also does not succeed if there is a fistula of the stomach and the colon (if a free passage exists), or it succeeds only after enormous quantities have been introduced. If one introduces water into the rectum, it reaches the stomach and is then sometimes vomited. When air is forced in through the anus, it first inflates the sigmoid flexure, then the stomach, and finally the ascending, transverse, and descending colon. After adhesions between the intestinal cancer and the front abdominal wall the subsequent disintegration of the same may form an external fistula of the intestines, which usually communicates with the outside through an irregular channel. Under such very rare conditions we may determine, by means of a careful chemical analysis and examination of the contents of the intestine, which exude through the fistula, whether they arise from the upper part of the bowel, the duodenum, jejunum, or ileum, or from the cecum or the colon. Histological evidences of cancerous tissue are also occasionally discovered under such conditions. Solutions of anilin colors poured into the stomach can be readily recognized on their prompt appearance in the rectum.

Course and Duration of the Disease.

There is much doubt concerning the duration, because we can not determine the date of the beginning of intestinal cancers. But when they are not complicated by other circumstances, the duration of the disease, so long as it gave rise to recognizable symptoms, has in some cases been estimated at four years; often, however, it is much less, while it may be considerably shortened by the occurrence of complications. Since the patient is subject to very many dangers due to the manifold complications and resulting circumstances of the intestinal cancer,—stenosis, hemorrhage, perforation and rupture, occlusion, autointoxication, direct extension of the cancer to other organs, as well as metastases in other organs, dilations of the stomach if the neoplasm is in the duodenum,—it is easily understood that the duration of the disease varies considerably in individual cases. Many patients may suffer early from coma carcinomatosum, which is attributed by some to autointoxication, although we do not yet know the toxic substances causing it. In cancer of the duodenum nutrition usually suffers early, because the cancer causes strictures of the intestine and stenosis of the pylorus. The gastric functions are severely disturbed, and the patients are unable to take up and digest a sufficient quantity of food. Cancer of the rectum is, as a rule, the one that can be endured for the longest time if complications do not arise, especially since the appetite remains undisturbed for a long time. Anemia resulting from an intestinal cancer may increase very quickly and be associated with thrombosis, or the patient may succumb to embolus of the lungs. In my personal experience intestinal cancer has never run its entire course without one or more of the complications mentioned, and it was by these that the fatal termination was directly brought about.

Diagnosis.

In the absence of tumor or enterostenosis, intestinal cancer can be suspected, but not diagnosed with certainty. But even if these signs are present, the existence of intestinal cancer is by no means proved, since the stricture may be caused by a cicatrix of a nonmalignant ulcer, and the tumor which is felt may be an intussusception or proceed from other abdominal organs. The early appearance of cachexia anemia, even when the nourishment is quite sufficient, is an evidence in favor of carcinoma, whereas in nonmalignant stricture the nourishment suffers only when the stricture causes disturbances in digestion or in the absorptive power of the intestine. The results of repeated examination of the evacuations (see Symptoms of Intestinal Stricture) are also of value for this differentiation. The latter applies also to the differential diagnosis of intestinal cancers and tumors of other abdominal organs. With reference to other differences, I refer to the diagnostic feature of tumors of abdominal organs in my work on "Diseases of the Stomach," second edition, pp. 556–558. If it is desired to learn more accurately the location of the stricture in intestinal cancers, it is advisable to introduce air or water into the intestine, and find out how far it penetrates. If there is a suspicion that the wall of the intestine is weak (perforation peritonitis), these methods are contraindicated.

Chapter xxxv, on Obstruction, and those on the various occlusions contain the diagnostic factors in reference to the localization of the various cancers of the intestines and their differentiation from fecal tumors; also the section on Symptomatology. If a swelling is found in the region of the duodenum, and if, after a while, continuous jaundice sets in, we may assume a cancer at the mouth of the common gall-duct. In differentiating between cancer of the pylorus and of the duodenum attention must be directed to ascertaining whether the contents of the stomach contain large amounts of free HCl or not when digestion is at its height. The characters of infra- and suprapapillary stenosis of the duodenum are given in the chapter on Enterostenosis.

Prognosis.

If the diagnosis of intestinal cancer is certain and an operation is not possible or the patient refuses to undergo one, the prognosis is very unfavorable, and one can not foretell how long life will last in various cases, since it may be shortened by unexpected events. The earlier the cancer of the intestine is recognized and the quicker it is extirpated, the more favorable are the conditions for a longer life and for the prevention of the metastases.

Therapeutics.

The life of the patient can be preserved or lengthened only by an operation. An artificial anus, gastro-enterostomy, and complete extirpation of the tumor are the surgical means employed, according to conditions found after laparotomy. In rectal neoplasms, if the cancer can not be operated upon, the complaints of the patient are at times ameliorated and the progress of the disease

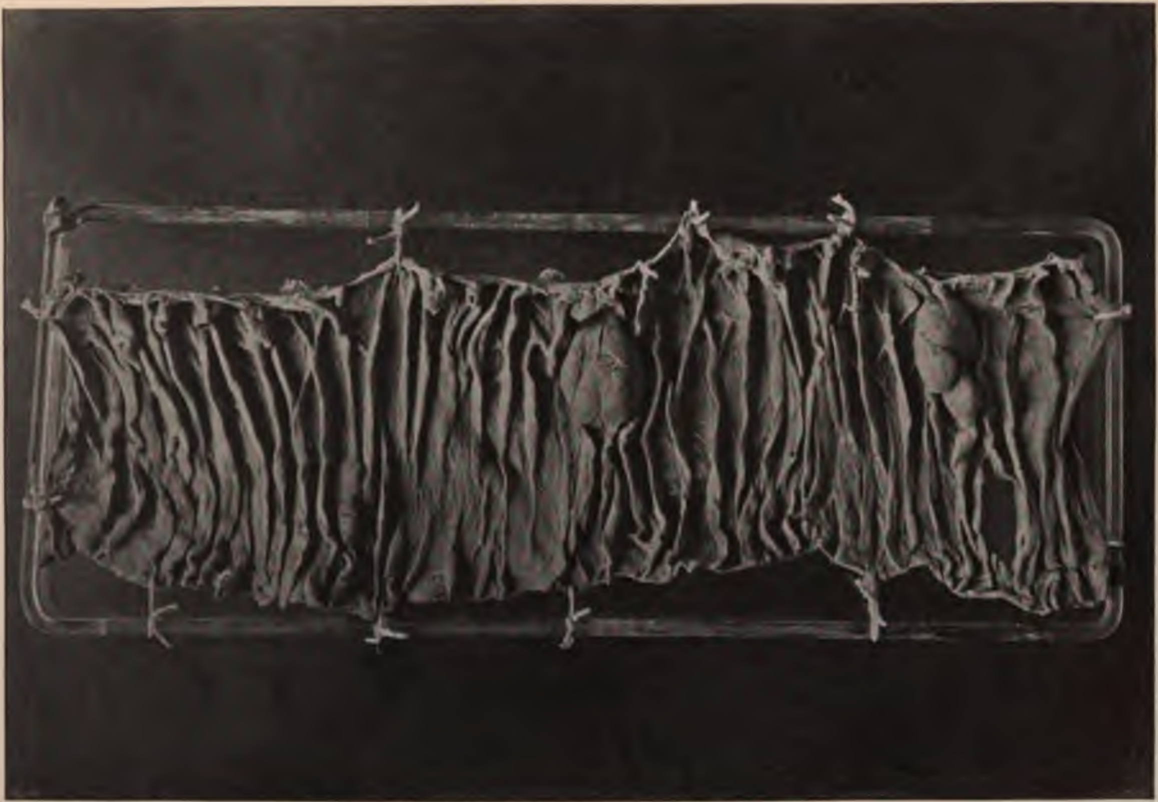


FIG. 38.—LYMPHOSARCOMATOUS NODULES IN INTESTINE.

somewhat checked by curetting out the neoplasm, in cancer of the rectum. If operation is refused or not feasible, symptomatic treatment only is possible—easily digested food (somatose, nutrose) in case of strictures situated in the colon. Nutrition should be carried on by nutritive enemata in case of duodenal or jejunal carcinomata. For violent pain, subcutaneous injections of morphin and also opium by suppositories should be given. If there are evidences that the stricture has reached an advanced stage, strong aperients must be strictly avoided, because they may cause perforation or rupture.

The treatment of cancer of the rectum has been given by Dr. Thomas Charles Martin in a special chapter. In fact, the entire treatment of intestinal carcinoma may be said to coincide with that of enterostenosis, or occlusion.

Besides cancers, other neoplasms occur, although more rarely, in the intestines—namely: sarcoma, polypi, lipoma, angioma, lymphangioma, cystoma, adenoma, and myoma.

SARCOMA AND LYMPHOSARCOMA OF THE INTESTINE.

These neoplasms involve the intestine extremely rarely, as is evident from the result of necropsies of large hospitals. Of 13,000 necropsies at the German University of Prague during fifteen years, there were only 13 cases of sarcoma of the intestine. This neoplasm takes its origin, as a rule, from the submucosa, involving the muscularis and serosa at an early date. There are, however, very rare cases reported in which the sarcoma seems to have arisen from the mucosa. They seem to have a predilection for the small intestine and the rectum, and are rare in the large intestine. The only case which I have observed, however, occurred in my service at the Maryland General Hospital. It was an enormous sarcoma of the splenic flexure of the colon, involving the transverse colon. At the autopsy the solid part of this neoplasm measured 19½ inches—about 42 cm.—in circumference. During life, owing to the accumulation of feces above it, it seemed much larger even than this. According to a report by F. R. Krüger (Inaug. Diss., Berlin, 1894) there were 37 cases of intestinal sarcoma up to that date. Since then I can find but 2 additional cases: one reported by C. van

Zwalenberg, reported to the Section on Surgery and Anatomy of the American Medical Association, fifty-first annual meeting, Atlantic City, June 5, 1900 (see "Phila. Med. Jour.," vol. v, p. 1276), which presented really two growths, involving the ileum and colon near the ileocecal valve, in a boy five years old, and a second case by Carrington (quoted by Baltzer, "Arch. f. klin. Chir.," Bd. XLIV, S. 744). Including these 2 in Krüger's 37 cases, the following topographical table can be arranged:

Small intestine,	16
Ileum and cecum,	2
Cecum,	2
Vermiform process,	1
Transverse colon,	1
Small intestine and colon,	1
Rectum,	16
Total,	39

From 1882 to 1893, during twelve years, there were 2125 autopsies on cancer cases in the Wiener allgemeines Krankenhaus. Of these, 243 occurred in the intestine. During the same period of twelve years 274 sarcomata were found, but only 3 sarcomata occurred in the intestine. Of 61 lymphosarcomata, only 9 occurred in the intestines. Nothnagel attributes importance to these figures because they were compiled by Kundrat, who was the director of the Pathological Institute and had given special attention to this subject. A similar relation was found by Müller at the University of Bern, where 41 out of 521 carcinomata had invaded the intestine, but of 102 sarcomata, only one had invaded the intestine.

The histological structure of the intestinal sarcoma is, in the majority of cases, that of a small round-cell sarcoma. But Krüger states that all possible forms have been observed—small round-celled, large round-celled, small spindle-celled, large spindle-celled also medullary, cysto-, and melanosarcomata. An excellent account of the histological structure of these neoplasms has been given by Ackermann ("Die Histogenesis und Histologie der Sarcoma," Volkmann's "Sammlung klin. Vorträge," 233, 234). While carcinomata, as a rule, produce a stenosis, or at least a narrowing of the intestine, sarcomata generally effect a dilation of

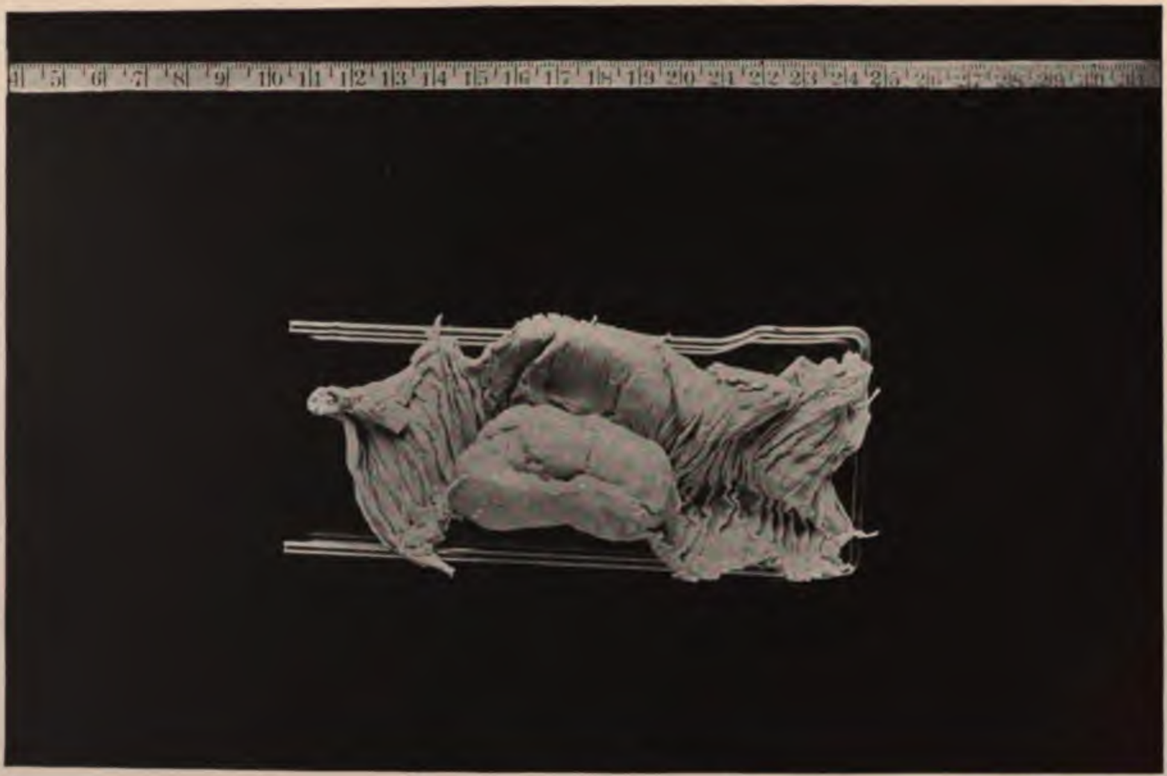


FIG. 39.—SECONDARY SARCOMA OF ILEUM.

the bowel. In the case of sarcoma of the splenic flexure of the colon observed by myself, the lumen of the bowel had been stenosed to a varying degree,—at some places the tortuous canal winding through the neoplasm was about half an inch wide; at others it dilated to an intraneoplastic ampulla about the size of a hen's egg. The canal had been absolutely stenosed by fecal concretions. In a case of lymphosarcomatosis reported by Rudolph Schmidt ("Wien. klin. Wochenschr.," May 26, 1898), the diagnosis of malignant stenosis of the intestine was made, but that of lymphosarcomatosis was excluded on account of the obvious existence of a stenosis. For Kundrat ("Wien. klin. Wochenschr.," 1893, No. 12) had asserted that this disease is usually accompanied by an expansion of the intestinal lumen. We do not command a sufficiently large number of clinical histories supported by autopsies to justify the adoption of this sign (expansion of the lumen) as a diagnostic factor.

Sarcomata are characterized by an enormous energy of growth, their general contour is smooth, and they frequently acquire large dimensions. The lymphosarcomata and sarcomata of the intestine are, as a rule, primary. They rarely extend to the intestine secondarily from other organs. They may extend to the intestine by contiguity, when lymphosarcomata exist primarily in the mesenteric or retroperitoneal glands.

The lymphosarcomata take their origin from the solitary, as well as the agminated, lymph-follicles, from which they may extend and involve very large districts of the intestine. Orth (*l. c.*, p. 847) describes them as being capable of involving the entire intestinal mucosa, so that it appears infiltrated with malignant lymphadenoma. While lymphosarcomata spread by invading adjacent lymph-follicles, the sarcomata may spread into any histological structure, and they exceed the carcinomata in the power of generating metastases.

Etiology.

What causes the growth of sarcoma and lymphosarcoma is not known. There is a great probability that they are of infectious origin. The close analogy between the clinical course of sarcoma and that of bone tuberculosis supports this view. There is also an interesting relation between tuberculosis and lymphosarcomatosis.

Rudolph Schmidt (*l. c.*), Dietrich (*l. c.*), Nothnagel (*l. c.*), A. Müller (*l. c.*), Claus (*l. c.*), and Ricker (*l. c.*) have referred to this connection, which, in some instances, appears more than a mere coincidence. Nothnagel (*l. c.*, p. 253) reports the case of a female patient, aged thirty-seven, in whom both an intestinal lymphosarcoma as well as intestinal tuberculosis were found by Weichselbaum at the necropsy. There were many cicatrices of tuberculous ulcers corresponding to the Peyer's patches or solitary follicles, and in the majority of these cicatrices grayish-white masses of neoplasm arose which, on microscopic examination, were proved to be lymphosarcomata. Rudolph Schmidt suggests that the coincidence of tuberculosis and lymphosarcomatosis is based on an inherited constitutional abnormality, expressing itself in a reduced power of resistance in the entire lymphatic apparatus. Wm. B. Coley ("Annals of Surgery," March, 1898) reports forty-six cases of sarcoma in which traumatism was regarded as the direct cause. This represents 30 per cent. of all the cases observed by this writer, who also regards sarcoma as of infectious origin.

Symptomatology.

The clinical history of these two varieties of neoplasms is characterized by a very early and grave reduction of the general vitality. Simultaneous with this the local symptoms are not predominant. Patients with intestinal sarcoma emaciate rapidly and are subject to anemia at an early date. Madelung ("Centralbl. f. Chir.," 1892, No. 30) asserts that this may occur when there are no symptoms pointing to the abdominal seat of the disease. There are no characteristic subjective symptoms. The symptoms of abdominal pain, nausea and vomiting, and alternating diarrhea and constipation are certainly not peculiar to these neoplasms. Treves (*l. c.*), Madelung (*l. c.*), Kundrat (*l. c.*), and Baltzer (*l. c.*) emphasize the absence of stenotic phenomena and the consequent absence of visible intestinal peristalsis and intestinal plasticity. Just the opposite is, as a rule, the case in carcinoma, which so often leads to enterostenosis. Wherever constipation is reported in the literature I noticed that it was due to an intussusception or enteroptosis. This observation I have found recorded in 7 cases out of 12 reported in literature in which any reference was made to these points. The extensive involvement of the muscularis by intestinal sarcomata and the consequent exclusion of peristaltic movements explained the expansion of the intestinal wall by the accumulating contents and the eventual dilation.

Tumor.

The tumor is, as a rule, smooth, movable, generally of a hard consistency, at times showing central foci of softening. It grows surprisingly rapidly. Madelung (*l. c.*) speaks of a rate of growth of such rapidity that its increase could be demonstrated in a few days. As shown in my case,—19½ inches at autopsy,—the tumors may reach enormous dimensions. Among the remaining symptoms which may occur, are edema of the feet and ankles and, occasionally, fever, which may reach about 39.5° C. The rare occurrence of intestinal hemorrhages in sarcomata does not impress me as being a valuable diagnostic sign, since there are cases of intestinal carcinoma in which no blood is found in the stools in the entire clinical history.

Age.

Of the 37 cases collected by Krüger, 22 occurred before the fortieth year.

Diagnosis.

Perhaps the most available diagnostic features are the absence of stenotic phenomena and the rapid growth of the neoplasm. The presence of tuberculosis simultaneously with an abdominal tumor of rapid growth does not necessarily mean that the latter is a lymphosarcoma. It might as well be a tubercular tumor. The diagnosis of lymphosarcoma meets with unsurmountable difficulties. In the case reported by Rudolph Schmidt (*l. c.*) a stenosis was present, and this led the clinician to exclude the possibility of sarcoma. In the absence of tumor the diagnosis becomes absolutely impossible.

Duration.

Most cases of intestinal sarcoma die within nine months. A few patients lived one year and nine months. One case died after two weeks. As the beginning of the neoplasm can not be determined with exactness, these figures can only be considered approximate.

Treatment.

The only hope of partial success is in surgical treatment. The case reported by C. Van Zwalenberg, an ileocecal sarcoma in a child five years old, recovered after the ascending colon and part of the ileum were excised, and an end-to-end anastomosis with a Murphy button was performed. In this case there had also been an intussusception of from three to four inches of the ileum. The child was well six months after the operation and had gained seventeen pounds. Engström (*l. c.*), Gilford (*l. c.*), Hahn (*l. c.*), and Michel (*l. c.*) have reported successful operations for sarcoma. Recurrences, however, are more frequently observed than in intestinal carcinomata. Whenever an operation is refused or impossible, Wm. B. Coley's treatment by injection of a mixture of the toxins of erysipelas and Bacillus prodigiosus may be tried ("Practitioner," April, 1899). Subcutaneous injection of arsenic has been employed by von Ziemssen (*l. c.*), and the results have been, on the whole, favorable, if we may judge from temporary symptomatic improvement.

BENIGN NEOPLASMS OF THE INTESTINES.

The benign neoplasms of the intestines present more of a pathological than of a clinical interest, for, excepting those occurring in the rectum, they are, in my experience of the literature of the subject, practically never diagnosed during the life of the patient. In the chapter on Enteritis I have described the conditions, under the term of enteritis polyposa and colitis polyposa, in which the mucous membrane of the small or large intestine may give rise to the formation of tumors varying from the size of a pea to that of a walnut. In the photographs represented in Figs. 41 and 42 two very characteristic intestines are graphically represented. They were kindly presented by Professor Wm. H. Welch, of the Johns Hopkins Hospital. Under specially unfortunate conditions, for instance, by inducing of an invagination, intestinal benign tumors may actually cause death. There is also a belief that a number of these neoplasms, particularly the adenomata, may be transformed into epitheliomata

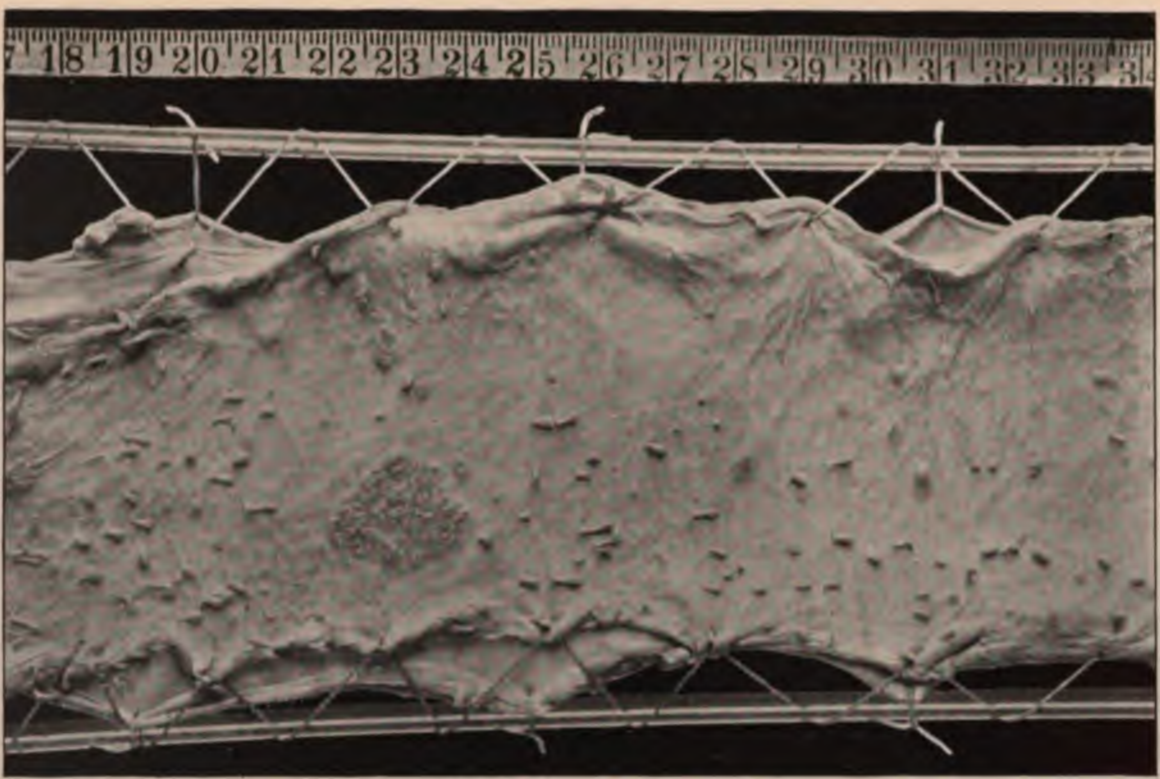


FIG. 40.—MULTIPLE POLYPI OF SMALL INTESTINE.

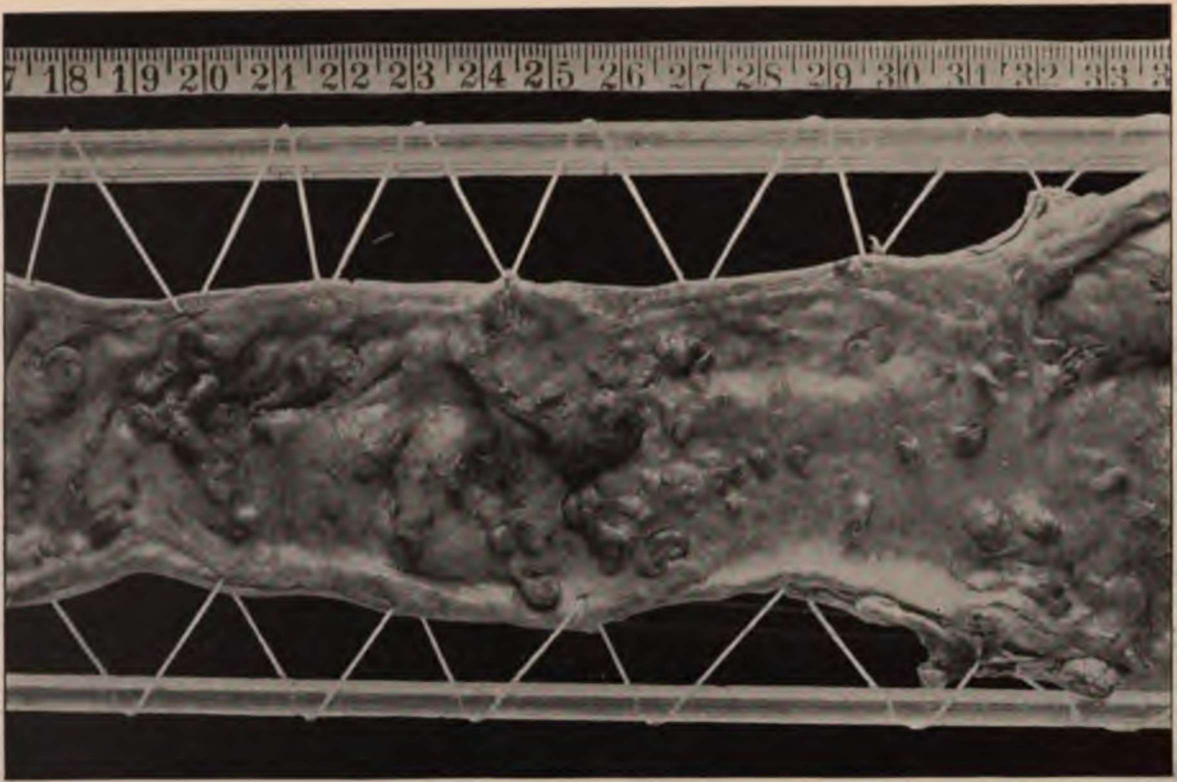


FIG. 41.—MUCOUS POLYPI OF COLON.

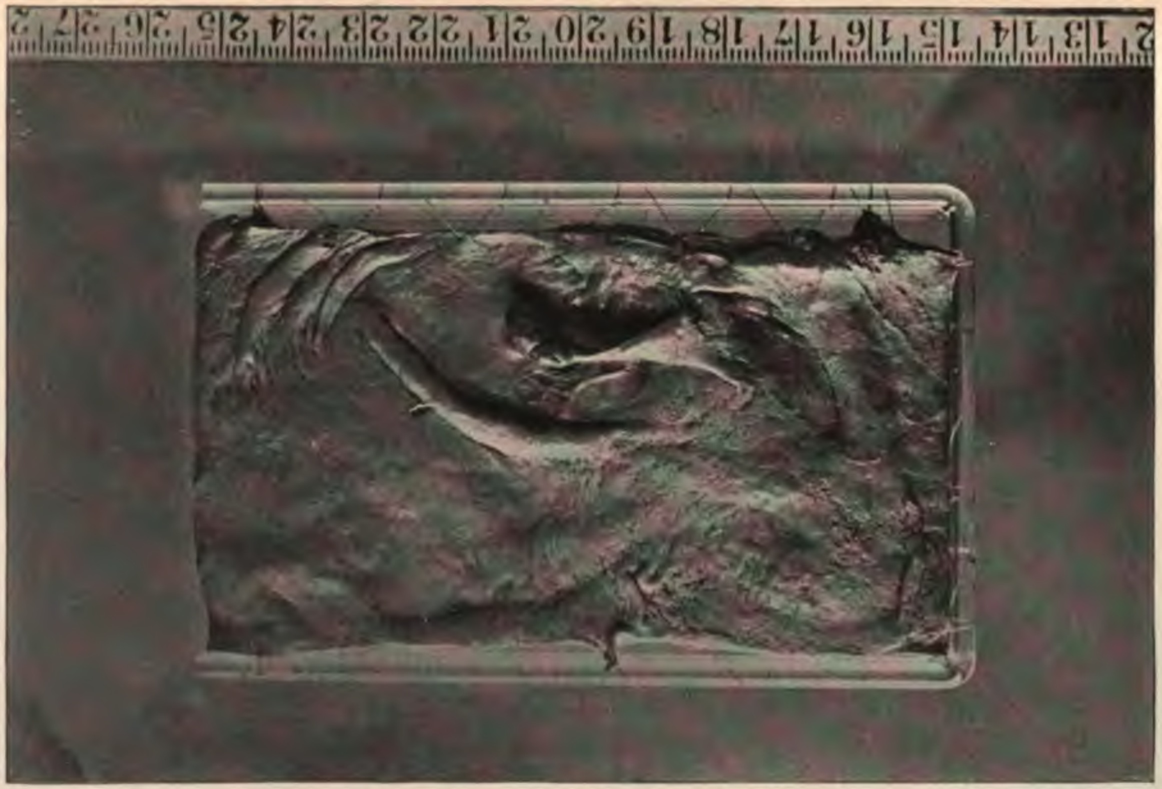


Fig. 42.—Polypoid Colitis (Colitis Polyposa).

(Hauser, "Deutsch. Arch. f. klin. Med.," Bd. LV, 1895, and Alb. Mathieu, "Traité des Maladies de l'estomac et de l'intestin," p. 928).

Terminology.

The term "intestinal polypi" embraces the whole group, but should not be used in that sense, because a polypus is generally understood to be a pedunculated neoplasm, or, at least, projecting from the surface on which it grows. Some of these neoplasms, however, are actually imbedded in the wall of the intestine. They are generally named according to the tissue from which they arise and which constitutes their main bulk. Thus the adenomata arise from the glandular tissue; the fibromata, lipomata, and papillomata, from the connective tissue; the myomata from the muscular tissue; the angiomata, from the blood-vessels.

Then there may be benign neoplasms of mixed tissue, known as myxomata. Finally, we may have a combination, or, what are, more correctly speaking, a transition of benign and malignant tumors—the myosarcoma, the fibrosarcoma, the myxosarcoma, etc.

Adenoma.

This type of benign neoplasm originates from the glands of Lieberkühn, and in the duodenum, from those of Brunner. They vary from the size of a pea to that of an orange, and may occur in all parts of the intestine, but especially in the rectum. They are said to occur most frequently during youth. The shape varies considerably. An adenoma may spread around the lumen of the intestine in the form of a ring, and cause a stenosis thereby. Adenomata may occur as flat elevations, as well as pedunculated, like polypi. One of the cases which I have pictured in the accompanying illustration (Fig. 40), exhibited a very large number of these polypi throughout the small intestine. They showed a glandular structure. This condition has been called by Hauser "adenomatous intestinal polyposis." Multiple polyposis of the large intestine of this sort has been described by Whitehead, Port, Schwab, Holtmann, and Luschka. About forty-two observations of this kind have been collected in a study by Quénu and Landal ("Revue de Chirurgie," April, 1899). Two of this collection are personal observations by these authors. Luschka estimated the number of polypi in his case at over 1000. Bardenheuer and Hauser had similar cases; in the case of the latter, however, the polypi extended from the pylorus to the rectum. Hauser found a carcinomatous degeneration in the polypoid growths.

In a highly neurasthenic man, forty-eight years of age, who had gone through several attacks of enterostenosis, which were generally ushered in by a period of constipation, absolute suppression of stool and the passage of flatus followed. In one of these attacks I had the opportunity to examine him. The transverse colon was enormously distended, and it was impossible to pass a Kuhn or Langdon tube further than the beginning of the sigmoid flexure. At first I made the diagnosis of volvulus of the sigmoid flexure, but the previous history—that he had had a dozen such attacks but they had passed over either spontaneously or by the use of copious injections into the rectum—made me hesitate as to the diagnosis. On examining the patient with one of the long Kelly proctoscopes at the University of Maryland I discovered a bunch of adenomatous neoplasms in the inferior curve of the sigmoid flexure. A number of them were removed very readily by curetting. The remaining portion had to be ligated and cut off. The operation was done on March 31, 1899, and in January, 1901, the patient has had no return of symptoms of enterostenosis. Careful inspection of the rectum is absolutely necessary in all cases where the stools are persistently mixed with blood and where there is tenesmus. Whenever there are multiple polypi in the rectum, it is very probable that similar growths are present further up in the bowel.

Lipoma, Fibroma, and Papilloma.

Lipomata originate from the submucosa, especially in the colon, sigmoid, and rectum. They may occur isolated or multiple, and vary from the size of a pin's head to that of a very large orange. Lipomata may, after having caused signs of enterostenosis, terminate spontaneously by sequestration and expulsion of the growth. These growths, which originate from the connective tissue, particularly of the submucosa, are exceedingly rare. The lipomata, when they do occur, are frequently found to have originated from the epiploic appendix projecting into the peritoneal cavity. Occasionally they have detached themselves from the pedicle, and have been

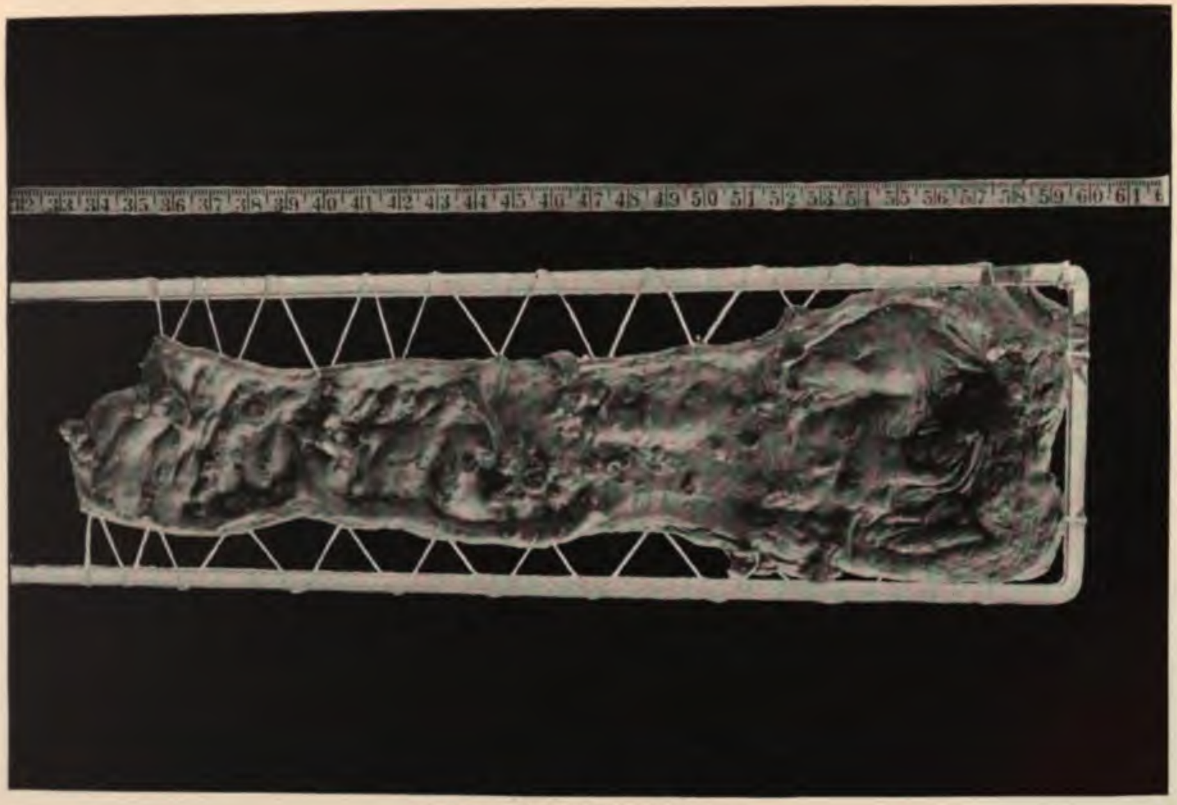


FIG. 43.—MUCOUS POLYPI OF COLON.

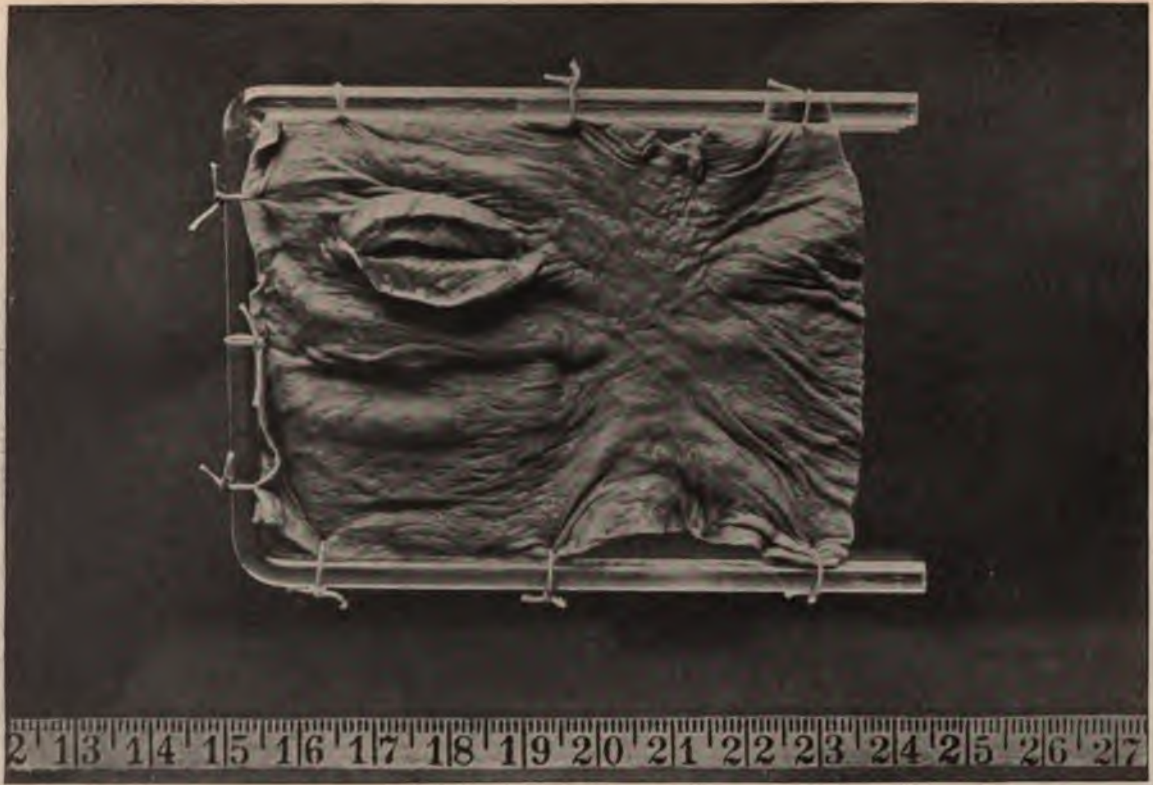


FIG. 44.—SUBMUCOUS LIPOMA OF COLON.

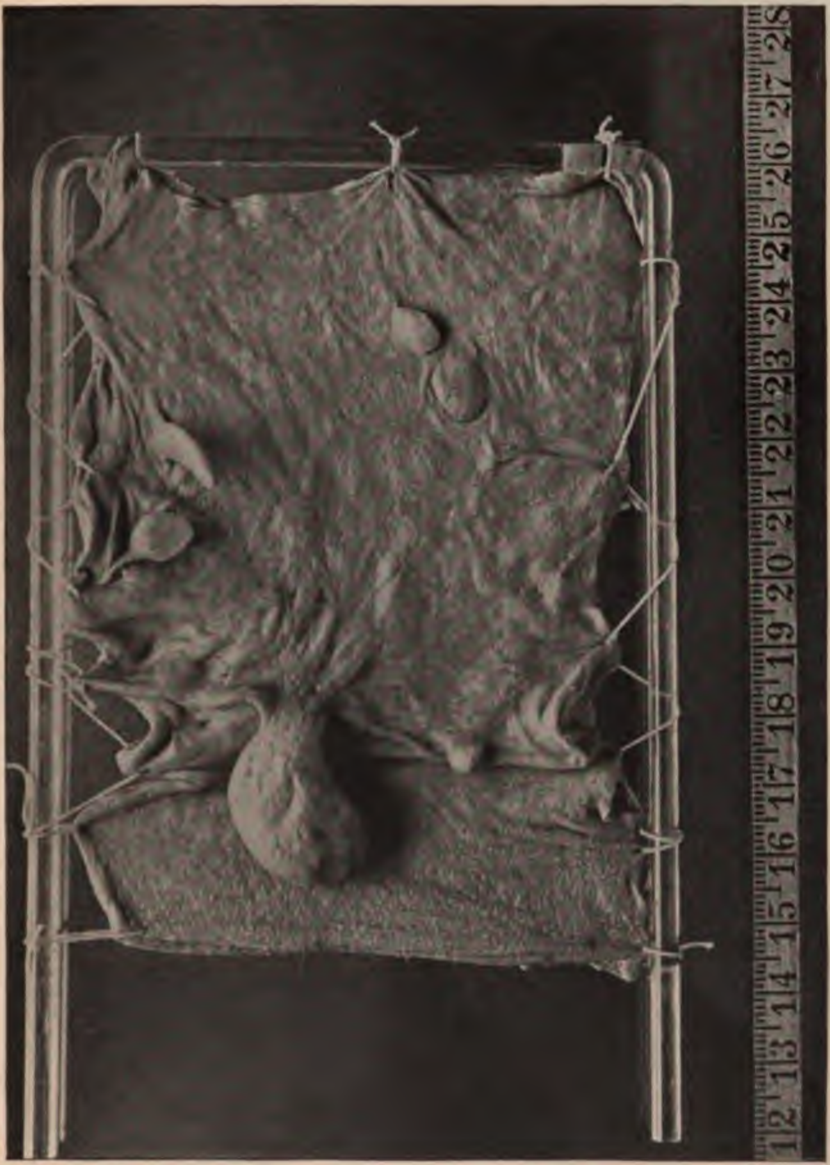


FIG. 45.—INTESTINAL POLYPI.

found free within the peritoneal space. The illustration (Fig. 44, opposite p. 718) from a specimen kindly presented by Professor Wm. H. Welch, of the Johns Hopkins University, represents a submucous lipoma of the colon.

Myomata and Fibromyomata.

Myomata may originate from the muscular coat proper or from the muscularis mucosæ. They are most frequently found in the small intestine and only exceptionally in the colon. The age of the patient may vary from early youth to the eightieth year. According to Boeticher ("Virchow's Archiv," Bd. CIV, 1886), and Lode ("Wien. klin. Wochenschr.," 1894, No. 21), there are two main types of intestinal myoma: (1) The first is a sharply circumscribed broad induration of the intestinal wall which originates from local hyperplasia of the longitudinal and circular muscular fibers. The mucosa and submucosa are also swollen and firmly adherent to the neoplasm. (2) The second form also originates by local proliferation of smooth muscle-fibers, but gradually assumes a spherical shape, does not become adherent with the mucosa, and then develops a pedicle. Some myomata do not grow toward the lumen of the intestine, but toward the peritoneal surface. Those that grow toward the lumen are called the "inner," and those that grow toward the peritoneum are called the "outer," myomata. Steiner (*l. c.*) has collected nineteen cases of the inner and sixteen cases of the outer type.

Symptoms of Benign Intestinal Neoplasms.

Unless the neoplasms are in the sigmoid flexure or rectum, or unless they acquire a size which can stenose the lumen of the intestine or produce an invagination, they run a latent course and are not discovered until the autopsy reveals them. There are no characteristic symptoms of this condition. About the only symptom of importance is that of intestinal hemorrhage, which is said to be significant if it occurs in entirely healthy individuals who have never had any intestinal symptoms before, and in whom no other demonstrable cause of enterorrhagia can be determined. Repeated copious intestinal hemorrhages of this kind, occurring in children, should especially suggest the presence of polypi. In intestinal polyposis we have the discharges of dysentery, but I do not con-

growing tumor which is nodular, tough, and not in con
the genitalia, producing a feeling of drawing pain w
movements are made upon it; (2) symptoms of imp
(3) intestinal hemorrhages.

Treatment.

Spontaneous cures of intestinal benign neoplasms,
lipomata and adenomata, have been observed (Hurteu
zarri). Von Karajan ("Wien. klin. Wochenschr.," 1
abstracted in "Phila. Med. Jour.," vol. III, p. 821) rep
of polyposis of the jejunum and ileum which had g
attacks of abdominal pain and distention. There w
appetite, vomiting of bile, and loss of ten kilograms in
patient gradually becoming worse, laparotomy was und
and one-half years after the first attack. On opening t
the loops of the small intestine were in five places f
trophied and distended above apparently stenosed are
lower ileum there was an invagination of five centimete
tip end of it a movable tumor the size of a nut was fou
centimeters further up, a second tumor. Still others were
higher up in the ileum, jejunum, and duodenum, but
was free. The intestinal wall was opened in five place
polypi of various sizes were removed. The patient made
recovery, and was well one year after the operation. Pro
of the University of Chicago, has operated successfull
called outer rectal myomata. Benign neoplasms highe

LITERATURE ON CARCINOMA OF THE INTESTINES.

1. Aaron, Chas. D., "Carcinoma of the Duodenum," "Phila. Med. Jour.," 1899, vol. III, No. 4, p. 280.

2. Albert, cit. nach Steiner (*l. c.*).

3. Albrecht, "St. Petersburg Med. Wochenschr.," 1880, No. 9.

4. Atkinson, I. E., "Cancer and Lardaceous Degeneration of the Intestines," "Pepper's Syst. Pract. of Med.," Philadelphia, 1885.

5. Baltzer, "Arch. f. klin. Chir.," Bd. XLIV, H. 4.

6. Bamberger, "Krankheiten des chylopoetischen systems," in "Virchow's spec. Pathologie und Therapie," Würzburg, 1864.

7. Barbes-Nanu, cit. nach Steiner (*l. c.*).

8. Bardeleben, "Carcinoma gelatinosum cœci et coli ascendentis," "Charité Annalen," 1883, Berlin, 1885, Bd. X, S. 423.

9. Bardenheur, "Arch. f. klin. Chir.," Bd. XLI, No. 4.

10. Barker, F., "Cases of Intestinal Cancer," "New York Medical Journal," 1883, vol. XXXVII, p. 64.

11. Bartholow, R., "Cancer of the Bowels," "Clinic," Cincinnati, 1875, vol. IX, p. 201.

12. Battle, W. H., "Cases of Resection of Large Intestine for Malignant Growths, etc.," "Lancet," No. 3976, November 11, 1899.

13. Behier, "Cancer de l'intestin," "Gaz. d. hôp. Par.," 1876, XLIX, 457, 474.

14. Bessel-Hagen, "Ein Ulcerôses Polyp des Jejunum bei einem Kinde," "Virchow's Archiv," Bd. XCIX, S. 99.

15. Bettelheim, K., "Ein Fall von Darmkrebs," "Med. und Chir. Rundschau," Wien, 1871, Bd. XII, S. 63–71.

16. Boas, "Carcinoma of Large Intestine," "Deutsch. med. Wochensch.r," 1900, 26. Jahrg., No. 7.

17. Boas, "Carcinoma of Large Intestine," "Deutsch. med. Wochenschr.," 1893, No. 41.

18. Bouley, "Epithelioma cylindrique de la valvulae ileocecale, obstruction intestinale," "Bull. Soc. Anat. de Par.," 1883, XI, 85–87.

19. Bristowe, J. S., "Cancerous and Other Growths of the Intestines," "Diseases of Intestines and Peritoneum," New York, 1879, pp. 61–64.

20. Brosche, "Zur Aetiologie der Carcinome des Verdauungstractus," "Wien. med. Wochenschr.," 1895, No. 40.

21. Browning, C., "Case of Secondary Malignant Disease Affecting the Ileocecal Valve and Obstructing the Intestine," "Brit. Med. Jour.," London, 1857, vol. I, p. 299.

22. Bryant, "Annals of Surgery," 1893.

23. Carwardine, T., "Spindle-cell Sarcoma of Rectum," "Brit. Med. Jour.," 1898, No. 1981.

24. Carwardine, T., "Excision of the Rectum for Cancer by a New Method," etc., "Brit. Med. Jour," No. 2026, 1899.

25. Carrington, von, "Arch. f. klin. Chir.," Bd. XLIV, S. 744.

26. Castell, du, "Cancer de l'ileon," "Arch. gen. de med.," Paris, 1882, II, 19–33.

27. Castelain, "Gaz. hebdom. de med. et Chir.," 1870, No. 20.

28. Chaboud, "Observation de tumeur carcinomateuse au niveau de la valvulae ileocecale," "Mem. et Compt. rend. Soc. d sc. med. de Lyon," 1874.1875, XIV, pt. 2, 107–109.

1856, vol. XVI, p. 270; also "Amer. Med. Monthly," New York, 18
also "New York Medical Times," 1855–56, vol. V, p. 244.

34. Cohnheim, "Vorlesungen über allgemeine Pathologie," Berlin

35. Crisp, E., "Villous Cancer (So called) of the Intestines," "T
Pathological Society of London," 1860–61, vol. XII, p. 113.

36. Czerny, cit. from Steiner (*l. c.*).

37. Dave, J., "Cancer de L'intestin generalisé au peritoine," "B
Par.," 1874, XLIX, 459–461.

38. Dejerine, J., "Estranglement interne par un epithelioma cy
intestin; noyaux secondaries dans le foie," *ibid.*, 1878, LIII, 288–292.

39. Descos and Beriel, "On a Case of Cancer of the Duodenum o
Form, Involving the Biliary Passages and the Head of the Pancreas,
Medecine," 19. me. Annee, No. 8.

40. Descroizilles, "Tumeur cancereuse de l'intestin grêle, du mese
epiploon; peritonite consecutive" (autopsie), "Bull. Soc. Anat. de Pa
23–29.

41. Dewey, "Case of Carcinoma of the Intestine," "Chicago
Exam.," 1878, vol. XXXVI, p. 346.

42. Dietrich, "Beiträge zur klin. Chir.," 1896, S. 377.

43. "Discussion sur un cas de degenerescence situe au niveau d
cecale," "Lyon Med.," 1873, XIII, 105.

44. Dunn, L. A., "A Case of Carcinoma of the Rectum," "
No. 3981.

45. Dupan, "De l'intervention chirurgicale dans le cancer du tub
rectum," Paris, 1883.

46. Engstrom, ref. nach "Arch. f. Verdauungs-Krankheiten," Bd.

47. Fenger, cit. from Steiner (*l. c.*).

48. Fenwick, "On the Atrophy of the Stomach," etc., London, 1

49. Ferguson, "Medullary Cancer of the Ileum Presenting Very Str
"Lancet," London, 1854, vol. II, pp. 378–397.

50. Finlayson, "Intestinal Obstruction," etc., "Glasgow Med. Jour
vol. XI, pp. 63, 317.

51. Fleiner, "Virchow's Archiv," 1885, Bd. CI, S. 484, u. f.

52. Fleur, "Étude clinique du cancer de l'intestin," Paris, 1879.

53. Galliet, "Cancer de l'intestin et du peritoine," "Bull. Soc. M
1874, XIII, 163.

57. Guerard, "Observations de squirrhe intestinal," "Bull. Soc. Anat. de Par.," 828, second edition, 1845, vol. III, pp. 165–167.

58. Hahn, "Berlin. klin. Wochenschr.," 1887, No. 25.

59. Hampeln, "Zur Symptomatologie occulter visceraler Carcinoma," "Zeitschr. f. klin. Med.," Berlin, 1884, Bd. VIII, S. 221–233.

60. Handfoot, "Transactions of the Pathological Society of London," 1890, vol. IV.

61. Hartung, "Scyrrhöse Verdickung des Magens und des Dickdarms," "Gen. Ber. d. k. rhein, Med. Coll.," 1840, Koblentz.

62. Hauser, "Das Cylinderepithel-Carcinom des Magens und des Dickdarms," Jena, 1890.

63. Hauser, "Deutsch. Arch. f. klin. Med.," Bd. LV, S. 429.

64. Haussman, "Contribution a l'history du cancer l'intestin ; etiologie et anatomie pathologique," Paris, 1882.

65. Heurteux, "Gaz. medic. de Nantes," 1884, S. 135 ; cit. nach Steiner (*l. c.*).

66. Hjelt, "Ett fall af medullart karcinom i tarmkanalen, Tinska lak sallsk. h. ndl.," Hellsingfors, 1879, XXI, 303.

67. Hollander, cit. from Steiner (*l. c.*).

68. Holtmann, "Multiple Polypen des Colon mit Gallertkrebs," Inaug. Diss., Kiel, 1895.

69. Homans, "Malignant Disease of the Large Intestine," etc., "Boston Med. and Surg. Jour.," 1861–62, vol. LXV, p. 125.

70. Johnson, "Resection of the Cecum and a Portion of the Ileum for Carcinoma," "Brit. Med. and Surg Jour.," vol. CXLI, No. 15.

71. Journet, "Étude sur le cancer de la terminaiason de l'intestin grêle," Paris, 1883.

72. Karajan, von, "Wien. klin. Wochenschr.," 1899, No. 6.

73. Keen, "The Advantages of a Permanent Abdominal Anus and of Total Closure of the Sacral End of the Rectum in Operations for Cancer of the Rectum," "Jour. Amer. Med. Assoc.," vol. XXXI, No. 7.

74. Kidd, "Cancerous Degeneration of Cecum and Commencement of the Colon," "Dublin Hospital Gazette," 1858, vol. V, p. 306.

75. Klebs, "Arch. f. exper. Path. u. Pharmakol.," Bd. I, 1873.

76. Kompe, "Zur Kentniss des Darmskrebses," "Aerztl. Intell. Bl ," München, 1883, Bd. XXX, S. 13, 26, 36.

77. Korte, "Phila. Med. Jour.," vol. V, No. 20, p. 1124.

78. Krausshold, "Ueber die Krankheiten des Processus vermiformis und des Cecum," Volkmann's "Sammlung klin. Vorträge," No. 191.

79. Krecke, "Adenocarcinoma of the Cecum," etc., "Münch. med. Wochenschr.," 46. Jahrg., No. 2.

80. Kruger, "Die primären Bindegewebsgeschwülste des Magendarmkanals," Inaug. Diss., Berlin, 1894.

81. Krukenberg, cit. from Steiner (*l. c.*).

82. Kukula, cit. from Steiner (*l. c.*).

83. Kundrat, "Wien. klin. Wochenschr.," 1893, No. 12.

84. Kuttner, "Multiple Carcinomatous Structures of the Intestine Produced Through Peritoneal Metastases," "Beiträge zur klin. Chir.," Bd. XXIII, H. 2.

85. Larsen, "Kankros striktur i Tyndt. ronen," "Norsk. Mag. f. Laegevidensk.," Christiania, 1877, 3, r, VII, 166–168.

86. Law, "Cancerous Disease of the Viscera," "Dublin Hospital Gazette," 1859, vol. VI, p. 123.

90. Leube, "Specielle diagnose der innerer Krankheiten," Le

91. Leudet, "Occlusion intestinal causé par un epithelioma sit pres de la valvule de Bauhin et consecutif a la dysenterie," "Bull. 1881, LVI, 21.

92. Link, "Wien. klin. Wochenschr.," 1882, S. 247.

93. Lockwood, cit. from Steiner (*l. c.*).

94. Lubarsch, "Ueber den primären Krebs des Ileum," etc., " Bd. CXI.

95. Luschka, "Ueber polypöse Vegetationen der gesammten Di "Virchow's Archiv," Bd. XX, 1861.

96. M'Cleilan, "Two Cases of Carcinoma of the Ileum," " Analect. Jour.," Philadelphia, 1824, vol. I, pp. 281–289.

97. McEwan, "Colectomy for Cancer of Hepatic Flexure," " Surg. Jour.," vol. II, No. I.

98. Madelung, "Centralbl. f. Chir," 1892, No. 30.

99. Makins, cit. nach Port (*l. c.*).

100. Marcacci, "Di un caso di occlusione intestinale per cancro, Firenze, 1872, XXIX, 244–249.

101. Maydl, "Ueber den Darmkrebs," Wien, 1883.

102. Michel, Inaug. Diss., Würzburg, 1889.

103. Mieville, de, "Cancer de l'intestin grêle," "Bull. Soc. Rom.," Lausanne, 1878, XII, 5.

104. Mohr, "Zur Geschichte der krebsigen Entartung des Darml schr. f. d. ges. Heilk.," Berlin, 1843, Bd. IX, S. 549.

105. Moutard, "Note sur un cas d'occlusio intestinæ," etc., "Fr 1877, XXIV, 641–643.

106. Moxin, "Cancer of the Small Intestine," "Transactions Society of London," 1872–73, vol. XXIV, pp. 641–643.

107. Muller, "Mittheilungen aus der med. Klin. zu Würzburg," Zürich, 1894.

108. Nothnagel, "Darmkrankheiten," S. 253.

109. Oliver, J. C., "Successful Resection of the Ileocecal Coi "Annals of Surgery," vol. XVII, No. 6.

110. Oser, "Primärer Dünndarmkrebs bei einem 25 jährigen Man Blätter," 1879, Bd. II, S. 33-39.

111. Paci, "Lo Sperimentale," 1882, S. 46; cit. nach Virchow-

116. Pfannenstiel, "Allgemeine med. Centralzeitung," 1897, S. 56.

117. Port, "Zeitschr. f. Chir.," Bd. XLII, H. 1 u. 2.

118. Powell, "Fatal Case of Carcinoma of the Ileum," etc., "London Med. Gaz.," 1850, vol. XI, p. 1029.

119. Prochownick, "Münch. med. Wochenschr.," 1896, No. 49.

120. Reeves, "Cancer of the Sigmoid Flexure of the Colon and First Part of the Rectum; Death from Perforation," "Assoc. Med. Jour.," London, 1853, 828.

121. Regeard, "Carcinome colloide de l'intestin grele," etc., "Bull. Soc. Anat. de Par.," 1877, LII, 473.

122. Reinhardt, "Charité Annalen," H. 1, Berlin, 1851.

123. Renonard, "Cancer miliaire de l'intestin et du grand epipsoon simulant l'occlusion intestinale," "Bull. Soc. anat. du Par.," 1879, LIV, 162–165.

124. Ricker, "Arch. f. klin. Chir.," 1895, Bd. L.

125. Ricketts, "Rectal Carcinoma with Subsequent Colotomy," "Phila. Med. Jour.," vol. IV, No. 7, p. 316.

126. Rosi, cit. nach Steiner (*l. c.*).

127. Ryall, "Extensive Transsacral Excision of Rectum for Cancer," "Brit. Med. Jour.," No. 2025.

128. Satterwaite, "Scirrhous Tumor of Ileum; Tortuous Condition of the Descending Colon," "New York Medical Journal," 1877, vol. XXVI, p. 533.

129. Schmidt, Rud., "Wien. klin. Wochenschr.," 1898, No. 21.

130. Schoering, "Ueber das Vorkommen des Mastdarmkrebses in den ersten beiden Lebensdecennien," "Deutsche Zeitschr. f. Chir.," Leipzig, 1885, Bd. XXII, S. 36–51.

131. Scholz, "Carcinoma of the Intestine—twenty cases," "Aerztl. Ber. d. k. k. allg. Krankenhaus zu Wien," 1880, 32.

132. Schuchardt, "Extirpation of a Cancerous Rectum and Sigmoid Flexure," "Deutsch. med. Wochenschr.," 25. Jahrg., No. 34.

133. Schuchardt, "Phila. Med. Jour.," vol. V, No. 20, p. 1124.

134. Schwab, "Beiträge zur klin. Chir.," Bd. XVIII.

135. Schweninger, E., "Darmkrebs Ann. d. stadt. allg. Krankenh. zu München," 1874–75, 1878, Bd. I, S. 369.

136. Seidler, "Krebs. des Dünndarms mit Durchfressung der Harnblase," "Med. Zeitung," Berlin, 1848, Bd. XVII, S. 210.

137. Senn, N., "Excision of High Rectal Carcinoma Without Sacral Resection," "Phila. Med. Jour.," vol. IV, No. 14, p. 607.

138. Sklifassowski, "Wratsch," 1881, No. 4; ref. nach "Centralbl. f. Chir.," 1881, S. 527.

139. Skoda, "Darmkrebs" "Allg. Wien. med. Zeitung," 1857, Bd. II, S. 83, 89, 93.

140. Smith, J. G., "Intestinal Cancer and its Treatment," "Lancet," No. 3908, 1898, "St. Bartholomew's Hospital Reports," 1887, vol. XXIII.

141. Sommer, "An Abdominosacral Method for the Removal of Rectal Carcinoma," "Med. Record," vol. LXI, No. 7.

142. Sorlin, "J. gen. de med., chir., et pharmacol.," Paris, 1824, LXXXVII, 180.

143. Southey, "Case of Intestinal Obstruction Due to Malignant Disease; autopsy, "Med. Times and Gaz.," London, 1875, Bd. II, S. 545.

144. Steiner, "Beiträge z. klin. Chir.," Bd. XXII, 1898, H. 1 u. 2.

145. Tarbell, "Colloid Cancer of the Intestine at the Ileocecal Valve," "Boston Med. and Surg. Jour.," 1878, vol. XCVIII.

148. Waldeyer, "Grosses Lipomyoma des mesenteriums," "Virchow's A
XXXII.

149. Waldeyer, "Die Entwicklung der Carcinome," "Virchow's Archi

150. Wallis, "Gastrojejunostomy for Pyloric Carcinoma," "Brit. Med.
1949, 1898.

151. Wardell, "Carcinoma of the Bowels," "Contrib. Path. and Pr
London, 1885, pp. 223, 232.

152. Weigert, "Ueber die Wege des Tuberkelgiftes zu den serösen
"Deutsch. med. Wochenschr.," 1883, No. 31 u. 32.

153. Westly, "Cancer of the Intestines with Secondary Deposits Elsew
"Liverpool Med. Chir. Journal," 1885, vol. v, pp. 470–472.

154. Whitehead, "Brit. Med. Jour.," 1884, vol. v, p. 410.

155. Whitties, "Transactions of Association of Amer. Physicians," 188

156. Ziemssen, von, "Deutsch. Arch. f. klin. Med," 1895, Bd. LVI, H. 1

157. Zwalenberg, "Phila. Med. Jour.," vol. v, No. 23, p. 1276.

INDEX OF AUTHORS.—VOL. I.

Compiled by the Author's Pupil, Dr. Charles C. Conser, Assistant Instructor Clinical Laboratory, University of Maryland.

A.

Abelmann, 66, 130, 418
Ackermann, 702
Acland, 623
Adler, 224
Afanassiew, 409
Akerlund, Sven, 488, 493, 499, 501
Albo, A. H., 48
Albu, 333, 336
Alexander, 551
Allen, 24, 35
Allingham, H., 371
Allingham, Wm., 371
Amerman, 115
Annesley, 482
Anrat, 63
Arnaud, 527
Askanazy, 652
Atwater, 74, 105, 106, 282, 287, 288, 317, 578
Aufrecht, 648
Ayres, 518

B.

Baginsky, 128, 480
Bamberger, 546, 658
Bardeleben, 103
Bardet, 336
Bardine, 618, 619
Baruch, S., 318
Basch, 89, 93, 249, 520
Bauer, 68, 305, 306
Baumann, 256, 413
Baumgarten, 638, 644
Bayle, G. L., 631
Beaumont, 335
Bedner, 413
Beer, A., 648
Beidert, 128, 464
Bellfrage, 478
Beneke, R., 670, 673, 674
Bergin, 242, 247
Bernard, C., 41, 54, 115
Bernstein, 54, 414
Berthelot, 42
Beschorner, 487
Bèvièl, L., 687
Bienstock, 161
Biernacky, E., 413
Billroth, 135, 630, 674
Birch-Hirschfeld, 648
Bird, Golding, 504
Bjornström, 648
Blackmore, A., 648
Blank, 42
Blondlot, 59
Boas, 42, 56, 214, 230, 232, 235, 237, 247, 256, 260, 261, 264, 303, 322, 329, 346, 371, 468, 470, 476, 487, 499, 501, 543, 596, 608, 651, 674
Boclo, 657
Boeck, 118
Boeticher, 723
Bokai, 93, 425
Bollinger, 631, 634
Booker, 103, 137, 165, 166, 422, 443, 536
Bouchard, 375, 376
Boucquoy, 602
Bourquelot, 38
Bowman, 253
Braam-Houkgeest, 84, 85
Branal, 63
Brett, 510
Brieger, 253, 255, 256, 321, 619
Brissaud, 614
Broder, T. J., 41
Brown, 48, 114
Brücke, 198
Brunn, C. de, 594
Bruno, G., 61
Bryant, A. P., 288, 614, 679
Bunge, 44, 52, 127, 245
Burwinkel, 595, 609
Busch, 38, 101, 196, 197, 198, 310

C.

Cabot, 180
Calmette, 527
Carrington, 702
Cash, 53, 75, 92
Cathcart, 224
Cavazanni, 66
Celli, 523, 524, 525, 527
Charon, 682
Chevalier, 489
Chittenden, H. H., 48, 114, 115, 261, 271, 410
Christomanos, 308

Cooley, Wm. B., 700, 708
Cordivilla, 616
Cordua, 47
Cornet, 638
Corney, 510
Councilman, 181, 192, 518. 522, 523, 528, 533. 537, 538, 556, 559
Courtois-Suffit, 488, 498, 588, 593, 624, 683
Cullen, Wm., 478
Cunningham, 522, 523
Curschmann, 32, 222
Cushing, 145, 147, 162, 163, 164, 199
Czerny, 69, 422

D.

Da Costa, J. M., 484, 487
Darras, 603
Dauber, 308, 487, 488
Davidson, 510
Deaver, 28
Demants, 36, 197
Demaschino, 560
Demme, 128
Demme, Karl, 331
Descos, 687
Devie, 608
De Waterville, 386
Dietrich, 706
Dieulafoy, 489, 651
Dobroklonsky, 637
Dock, 221, 538
Duckworth, Sir Dyce, 489, 490
Duclaux, 42
Dunin, 362, 376
Durham, H. E., 438

E.

Eberth, 171, 625
Ebstine, 652
Edebohls, 217
Edkins, 62, 63
Exner, 85, 99

F

Fellner, 90
Fenwick, 372, 594, 6
Fernelius, 484
Finger, 552, 658
Finkler, 187
Fiocca, 523, 524, 525
Fischer, 637
Fischl, 251, 449
Fitz, 372
Fleiner, 362, 503
Fleischel, 46, 75
Fleischer, 41, 45, 49, 113, 115, 120, 2 410, 412, 605
Flexner, 168, 169, 1 537, 538
Flügge, 192
Fontanus, Jacob, 594
Forster, 648
Fortzer, 619
Fosske, 404
Foster, 71
Fraentzel, 224
Francke, 504
Fränkel, 190
Frerichs, 232, 413, 59 658, 674
Frisby, 288
Fürbringer, 224

G.

Gad, 43, 84
Gaffy, 171, 174
Gairdner, 38
Galen, 515
Galliard, 506
Garland, 38
Garrod, Sir A., 489, 49
Gartner, 437, 439
Gaylord, H.

Grassi, 522, 523
Grawitz, 629
Greenwood, 617
Griesinger, 582
Grube, 429
Gruber, 65
Grünert, 38
Grützner, 99, 100, 300, 308, 311

H.

Hahn, 648, 708
Haig, Alex., 489, 490
Haliburton, W. D., 41
Haller, A. von, 412
Hammarsten, 197
Hankin, 181
Hansemann, 673
Hauser, 673, 679, 680, 681, 715
Hayem, 489, 490
Hedenius, 478
Heidenhain, 41, 49, 50, 54, 64, 73, 79, 417
Heimann, Geo., 680, 681
Hemmeter, 40, 69, 78, 119, 126, 130, 196, 210, 218, 224, 247, 251, 259, 262, 264, 295, 299, 303, 339, 387, 392, 399, 408, 414, 415, 419, 422, 423, 426, 601, 610, 647
Henoch, 422, 614
Herter, C. A., 375, 376, 377
Herxheimer, 631, 632
Heryng, 211, 212
Herz, Hans, 439, 542, 544, 630, 651
Heubner, 652, 658
Hicks, 91
Hildebrand, 224
Hirsch, A., 518, 582
Hiss, 182
Hodder, 314
Hodge, C. F., 348
Hoffa, 385
Hoffmann, 224, 560
Hoffmeister 62, 73, 243, 410
Holtz, 331, 332
Honig, 632, 638
Hoover, C. F., 556
Hoppe-Seiler, 50, 232, 418
Houze, 603
Howard, Jr., Wm. T., 523, 538, 556
Huber, 306
Hunter, 617, 618

I.

Illoway, 366, 367, 372, 374, 380, 385, 387, 628
Israel, 252
Issaeff, 185

J.

Jacoby, 330
Jacusiel, 481
Jaffé, 47, 253, 254
Jaksch, von, 240, 243, 244, 247, 249, 254, 255, 257, 332, 333, 439
Jaworski, 260, 326, 476
Johnson, Henry J., 650
Jolles, 48
Jolly, 40

K.

Karajan, von, 74
Karst, 315
Kartulis, 191, 193, 519, 522, 524, 525, 527, 528, 533, 534, 536, 541, 555, 559, 561
Kast, 315
Kelly, H. A., 378
Kelsey, C. B., 371, 378
Kersberger, 249
Kijanitzin, 429, 436, 437, 619
Kitagawa, 486, 497, 499
Kjelberg, 250, 251
Klein, 190
Koch, 183, 185, 186
Kocher, 224
Koebler, 251
Kölliker, 37, 70, 409
Koranyi, F. von, 629
Körte, 224
Kortum, 655
Kovacs, 533
Kraske, 681
Krause, J., 594, 601, 602, 619
Krueg, 315
Kruger, F. R., 701
Kruse, 193, 525, 528, 533
Krysinski, 486, 488, 494
Kuber, 181
Kuhn, 50, 267, 268, 463, 716
Kühne, 70, 71, 114, 210, 252
Kundrat, 703
Kupffer, 89
Kussmaul, 87, 503, 623
Kuster, 224
Kustner, 374
Kyber, 625

L.

Lafleur, 191, 192, 193, 523, 528, 533, 537, 538, 556, 559
Lambl, 520, 522
Landenberger, 315
Landois, 90, 94, 101, 409
Landweih, 42
Lange, 616
Langenhagen, 488
Langermann, 464
Langhans, 47

Leichtenstern, 236, 239, 363, 364, 365
Lennander, 614
Leo, 220, 246, 247
Lépine, R., 414, 417
Lesage, 142, 164
Letcheff, 488
Leube, 37, 120, 254, 259, 303, 304, 305, 315, 377, 488, 608, 626, 631, 659
Leubuscher, 64, 385
Leuwenhoek, Van, 135
Levin, 199
Levy-Dorn, 214
Lewin, L., 490
Lewis, 521, 522
Leyden, 222, 485, 488
Litten, 486, 498, 652
Livingood, 162, 163, 164, 199
Lockwood, 614
Loesch, 520, 522
Long, 617
Longuet, 486
Lorenz, H., 257, 623
Lösch, 191, 193
Löwenstein, 487
Lubarsch, 438
Lüderitz, 84
Ludwig, 73, 89
Lukjanow, 261, 263, 405, 412, 416, 420
Luttgen, 331, 332
Lutz, 521, 523

M.

Macfadyen, 50
Mackenzie, 614
Madelung, 706, 707
Maier, 623
Maixner, 252
Mall, F. P., 20, 91, 92, 93, 97, 98, 311
Mallory, 182, 521
Maly, 49, 412
Maragliano, 433

Mendelson, W., 4
Menzel, 315
Mering, 12, 63, 41
Michaelis, 257
Miller, 136
Miller, Casper O.,
Minich, 246
Minkowski, 47, 66,
Mitchell, Weir. 560
Moore, 78, 242, 24
Morax, 256
Morgagni, 484
Moritz, 120, 281, 2
Morot, 595, 600
Morris, 114
Mosler, 45
Müller, Fr., 42, 50,
132, 256, 409,
Munk, J., 49, 76, 7
Murchison, 605
Musculus, 65

Nasse, 50, 89
Naunyn, 47
Neilson, 498
Nencke, 42, 50, 75,
Nesbitt, 337
Neumeister, 48, 73
Nickel, 651
Noorden, von, 256,
Normand, 522, 527
Nothnagel, 84, 85, 8
97, 99, 100, 103
234, 238, 245,
309, 330, 347,
371, 376, 377,
459, 461, 462,
497, 498, 499,
617, 622, 626,
655, 658, 659,

Orth, 621, 633, 638, 644, 681, 682, 703
Ortweiller, 254, 255
Oser, 419, 648
Osler, 228, 486, 518, 522, 538, 589, 692
Osterben, 545
Overbeck, 482

P.

Pagenstecher, 612
Parenski, 622
Pariser, 240
Pasquale, 193, 525, 528, 533
Pawlow, J. P., 41, 43, 57, 59, 109, 115, 126, 134, 272, 414, 417, 420, 464
Peale, Albert C., 316
Peck, C. H., 294
Penzoldt, 116, 223, 609, 643
Perco, 315
Perier, 374
Perry, 615
Petruschky, 661
Pettenkoffer, 185
Pewlaski, 260
Pfaff, Franz, 549
Pfieffer, 174, 175, 177, 186
Pflüger, 89
Pfunger, von, 375
Pick, 219
Piorkowski, 183
Pitfield, 172
Pocanowski, 252
Poelchen, 651
Ponfick, 619
Pontain, 487
Praussnitz, 118
Prior, 187
Pröscher, F., 252

Q.

Quènu and Landl, 715
Quincke, 38, 47, 232, 233, 523, 548

R.

Rachford, 261, 410, 411
Ramdohr, 387
Ramm, 329
Ranke, J , 74
Rasch, 561
Reckmann, 602, 608
Recklinghausen, von, 418
Reichmann, 211 212
Reid, E. W., 66
Ribbert, 222, 673
Richardson, 183
Riedel, 221, 648
Rieder, 243, 648
Riegel, 100, 259, 306
Roberts, Sir Wm., 65
Robin, 47, 66, 608
Robson, A. W. M., 419, 420
Rodziejewsky, 102, 252
Röhmann, 49, 106, 127, 409
Rokitansky, 206, 570, 626
Rolle, 185
Roos, 233, 253
Roosbach, 54, 482
Rosenbach, Jung, 254
Rosenheim, 310, 426, 488
Rosenthal, 224, 303
Rothmann, M., 492, 494
Rothmann, O., 485, 492, 499
Roux, 608
Rovighi, 336
Ruber, 78, 105, 113, 118, 276, 382

S.

Salkowski, 47, 243, 253, 256
Salvioli, 73, 94
Schattenfroh, 190
Schierbeck, N. P., 398
Schiff, 37, 130, 197
Schillbach, 92
Schliep, 609
Schmidt, 234, 235, 248, 249, 499, 502, 543, 703, 706
Schmidt-Mülheim, 70, 71, 73
Schmitz, 336
Schmulewikch, 41
Schorlemmer, R., 52, 244, 245
Schottelius, 184, 423, 424, 425
Schuberg, 523
Schultz, Max, 619
Schutz, 339, 403
Schwann, 116
Sèe, G., 487, 499, 502
Seiber, 50
Semmola, 386
Shaw, 605
Shield, 614
Shiga, 167, 168, 169, 537
Shore, 73
Sigaud, 510
Silberschmidt, 167
Silvestri, M. de, 525
Singer, 251
Smith, E. E., 375, 377
Smith, Theobald, 139, 140, 143, 144
Snyder, 288
Southgate, 410
Spiegelberg, 598
Stadelmann, 51
Starke, 602
Sternberg, 181
Stich, 604
Stienach, 90
Stiller, 251, 449
Stolnikow, 416
Stomyer, 252, 660

Strümpell, 118
Sucksdorff, 136
Swartz, 595, 696
Swiezynski, 308

T.

Tacke, 397
Talma, 205
Tamman, 114
Tappeiner, 134
Tchitschowisch, 637
Thankhoffer, 50
Thayer, 538
Thierfelder, 196, 199, 423
Thiry, 37, 102, 197
Thiry, Vella, 37, 66
Thompson, Gilman, 574
Tiedmann, 61, 62
Tiersch, 674
Treaves, 18
Tremaine, 224
Trietz, 652
Trousseau, 482
Turck, 210, 272, 345

U.

Uffelmann, 105
Ullmann, 486, 552

V.

Vamossy, J. von, 330
Vanni, 487, 491, 493
Veillon, 190
Vidal, E., 340
Vierordt, 232
Virchow, 47, 363, 371, 376, 482, 558, 674
Voetsch, 363
Voight, von, 41, 357
Voit, 50, 66, 68, 77, 106, 117, 127,
305, 306, 409
Vonwyl, 607

W.

Warfinger, 605
Webster, Noah, 518
Wegscheider, 242
Wehgsarg, 241
Weir, 603, 604, 605, 611, 612, 613, 616
Weiske, 118
Welch, 187, 189, 197, 420, 619
Wenz, 197
Wertheim, 619
Westbrook, 179
Westphalen, H., 377, 379, 387
White, Hale, 504, 623
Whittaker, 315
Wilson, J. A., 24
Wistinghausen, 49
Woodruff, C. E., 577
Woodward, 357, 444, 478, 481, 484, 485,
518, 519, 522, 528, 534, 535, 537,
541, 549, 553, 554, 555, 559, 560,
566, 570, 589, 626, 631, 644, 658
Wüderhofer, 633
Wyss, 631

Z.

Zabludowski, Z., 385
Zeihl, 172
Zemann, 680, 681
Ziemssen, 225, 708
Zuber, 190
Zuntz, 680
Zwalenberg, C. Van, 702, 708
Zweifel, 52

INDEX OF SUBJECTS.—VOL. I.

Compiled by the Author's Pupil, Dr. Wilbur F. Skillman, Instructor in Clinical Laboratory, University of Maryland.

A.

Abdomen: changes in form of, 206; diminution in size of, 209; irregular enlargement of, 208; uniform enlargement of, 207
Absorption: paths of, 63; of water, 63; of salts and soluble constituents, 64; of carbohydrates, 65; of proteids, 68; changes in albumose and peptone during, 72; of fats, 74; of fats, emulsion theory of, 78; of fats, solution theory of, 79
Acetone, 257
Acholia, 412
Acholic stools, 232, 245
Achylia pancreatica, 416
Adenoma, 715
Albumin in feces, 242
Albuminuria, 251
Albumoses and peptones: in feces, 243; in urine, 252
Amebæ coli, 191, 520; biological properties, 191; lesions produced by, 192; reproduction in animals, 193
Amyloid ulcerations, 624
Amylopsin, 41
Antidiarrheic medicines, 324; contraindications to, 328
Antiseptics, intestinal, 98, 332, 339
Anus, as portal for infection, 509
Aperistalsis, 84
Atony, intestinal, 376
Auscultation, 221
Autointoxication, intestinal, 332; treatment of, 337

B.

Bacilli, imperfectly described forms, 161
Bacillus: aërogenes capsulatus, 187; morphology, 187; cultural properties, 188; fermentation formula, 188; pathogenesis, 189; bacteria resembling, 189
Bacillus: botulinus, 170; pathogenic properties, 171
  butyricus, 160; staining reaction, 160; cultural properties, 160
  coli communis, 137; morphology, 138; staining properties, 138; cultural characteristics, 138; indol reaction, 139; pathogenic properties, 141; agglutination reaction, 142; fermentation formula of, 143
  dysentericus, 167, 537; agglutinative reaction, 168; pathogenic properties, 168; Flexner's work on, 168; different from typhoid, 169
  fecalis alcaligenes, 161
  Gärtner, 148
  lactis aërogenes, 142; resemblance to colon bacillus, 142; pathogenesis, 143
  O and Bacillus paracolon, (Gwyn), 159
  proteus vulgaris, 165
  putrificus coli, 161
  pyocyaneus, 169; cultural properties, 169
  subtilis, 159; cultural properties, 159; ferments formed, 160
  typhosus, 148, 171; morphology, 171; stain for flagella, 172; cultural characteristics, 172; differential diagnosis from B. coli communis, 173; pathogenesis, 173; isolation from intestine, 182
Bacteria, intestinal, 135; first observations

on, 135; number of, 136; difficulties in study of, 163
Baths, 344
Bile, 45; antizymotic influence of, 413; acids, 51; chemical reaction of, 47; cholesterin of, 47; constituents, 45; fate of constituents, 51; constituents in feces, 244; disturbances in formation and flow of, 125; diastatic ferment of, 47; obstructed flow of, 126; pigments, 47; fate of pigments, 51; salts of, 46; tests for, 244; uses and functions of, 48
Blastomycetes, 162
Blood in feces, 235, 240, 243, 655

C.

Calculi, pancreatic, 236, 246
Cancer, intestinal, 672; course and duration of, 696; diagnosis of, 697; duodenal, 694; etiology of, 672; fever in, 695; fistula in, 696; location of, 679; metastases in, 681; occurrence of tumor in, 690; pathological anatomy of, 683; prognosis, 698; rectal, 692; intestinal, stricture in, 687; stool in, 688; symptomatology, 686; therapeutics, 698; types of, 678
Carcinoma intestinale, 670
Casein in feces, 239
Cathartic diarrhea, 351
Cecum, 28
Chemistry of feces, 241
Chlorosis, 374
Cholera: immunity against, 186; Widal reaction in, 186
Colic, intestinal, 394; diagnosis and treatment, 396; etiology, 394; fecal, 373; mucous, 483; symptomatology, 395
Colitis: acute, 452; ulcerative, 479; membranous, 483; definition, 483; nature and concept, 485; etiology, 487; pathology, 491; examination of intestinal discharges in, 495; comparison with similar processes, 498; complications, 498; symptomatology, 499; diagnosis and prognosis, 500; treatment, 500; diet in, 502; irrigation of colon in, 503
Colon: abnormalities of position, 31; ascending, 30; lavage of, in dysentery, 585; splenic flexure of, 31; transverse, 30; test lavage of, 463
Colon bacillus, fermentation, formula of, 143
Colon group, 137
Compresses, 343
Concretions in feces, 236
Constipation, habitual: diet for, 296; treatment with mineral waters, 298
Constipation—coprostasis, 356
Contraction, tetanic, of intestine, 97
Convalescence, diet for, 300
Coproliths, 218, 372
Coprostasic fever, 374
Crystals in feces, 239
Culture methods, 137
Cylinduria, 251
Cysts, pancreatic, dangers of puncturing, 224

D.

Decubital ulcer, 628
Defecation, 101
Diacetic acid, 257
Diarrhea, 350; etiology, 350; dyspeptic and dystryptic, 351; cathartic, 351; stercoral, 351; entozoic, 352; nervous, 352; after taking cold, 353; from irritants from blood, 353; treatment of, 353; diet for, 299, 300, 354
Diazo reaction, 256
Dietary prophylaxis and hygiene, 288; special, 299
Diet during epidemics, 288
Dietetic treatment, principles of, 273
Digestion: behavior of intestine during, 107; influence of bacteria upon, 194
Digestive secretions, special food excitants of, 59
Diphtheritic dysentery, 479
Douches, 343
Duodenal test-meals, 414
intubation: Hemmeter's method, 264; apparatus for, 264; Kuhn's method, 267
ulcer, 594; digestion, effect of gastric anacidity and hyperacidity upon, 58
Duodenitis, 451
Duodenum, 22; cancer of, 604
Dysentery, 515; classification, 517; geographic distribution, 517; etiology, 519; pathologic histology, 527; Japanese and Philippine, 536; symptomato-

logy, 539; evacuations of, 541; tormina and tenesmus, 550; urine in, 552; complications in, 553; peritonitis in, 553; intussusception in, 554; abscess of liver in, 555; nerve complications in, 559; symptomatology of rarer forms, 561; prognosis, 565; diet for, 299, 300; diphtheric, 479; chronic, 565; pathology of chronic, 569; ulcers of chronic, 570; treatment of, 574; disinfection of feces in, 581; dietetic treatment in, 581; colon lavage in, 585; medicines in, 589; after-treatment of, 591
Dystrypsia: bill of fare for, 293; treatment by mineral water, 295

E.

Electricity, treatment by, 345; indications for, 348
Electrodiaphany, 210
Embolic and thrombotic ulcers, 620
Emulsification of fats by pancreatic juice, 43
Enemata: absorption and digestion of, 100; preparation of, 311
Enteritis: acute, 433; accompanying infectious diseases, 439; after colds, 437; after burns, 436; course and extent of, 450; diagnosis, 455; etiology, 435; fever in, 449; influence of seasons on, 440; influence of age and sex on, 440; infections, 437; localization of the catarrh, 451; nature and conception, 433; pathology, 441; prognosis, 455; secondary or symptomatic, 438; symptomatology, 444; therapeutics, 456
  chronic, 458; clinical types of, 462; diet lists for, 471; dietetics of, 467; hydrotherapy in, 472; medicines in, 474; pathology, 458; prognosis, 465; symptomatology and diagnosis, 460; treatment, 466
  diphtheric, croupous, or pseudomembranous, 479
Enteritis: diphtheric, classification, 480; due to mercury, 481; due to constipation, 482; secondary, 483
  follicularis, 627
  membranous, 483
  phlegmonous, 477; etiology, 478; symptomatology and treatment, 479
Enterodynia, 394
Enteroliths, 236
Entozoic diarrhea, 352
Epidemics, food forbidden during, 292
Epithelial cells in feces, 240
Etherial sulphates, 255
Extrabuccal feeding, 301

F.

Fat in feces, 238, 250
Fecal colic, 373
Feces: amount of, 231; albumin in, 242; albumoses and peptones in, 243; bile constituents in, 244; blood in, 235, 240, 243; concretions of, 236; color of, 232; consistency and form, 231; casein in, 239; crystals in, 239; chemistry of, 241; examination of, 229; epithelial cells in, 240; fat in, 238, 250; fermentation of, 247; food particles in, 233; fragments of growths in, 237; fetal, 135; macroscopic examination of, 233; microscopic examination of, 237; muscle-fibers in, 237; mucin in, 242; mucus in, 234, 240; odor of, 233; pus in, 236, 240; phenol, indol, and skatol in, 249; proteids in, 250; reaction of, 241; starch in, 238; unorganized ferments in, 246
Ferments: in colon and rectum, 309; in feces, 246
Fibroma, 716
Fibromyomata, 723
Fistula of stomach and colon, 696
Flatulence, agents for relief of, 329
Flora, intestinal, 136
Fluctuation, 218
Follicular ulceration, 627
Food: amount of, 106; composition of, 106; digestibility of various kinds of, 110; kinds of, 105; preparation of, 107, 120; utilization of, by stomach and intestines of healthy man, 105; utilization of, in intestines under abnormal conditions, 108

Gastro-intestinal canal, specific irritability of, 59
Gout, intestinal ulcers in, 651
Growths, fragments in feces, 237

H.

Hemorrhagic pancreatitis, 420
Hemorrhoids, diet for, 296
Hog cholera, or intermediate group, 145; fermentation formula, 146; pathogenic properties, 147; general immunity, 147
Hydriatic treatment, 342; cases adapted for, 344
Hypernutrition, results of, 121
Hypochondriasis in obstipation, 376
Hypochylia, 416
Hypodermic feeding, 314

I.

Ileocecal valve, 27
Ileum, 24
Indican, test for, 253
Indicanuria, 252
Indirubin, 255
Indol, 249; reaction, 139
Infection, by anus, 509
Inflation of intestines, 225; by air, 225; by water, 228
Inspection, 205
Intestinal indigestion, 405; bill of fare for, 293; treatment by mineral water, 295
  colic, 394; ulcers, 592; neoplasms, 672
Intestines: physiology of, 36; peristalsis of, 84, innervation of, 89
Intravascular feeding, 314
Intubation, duodenal: Hemmeter's method, 264; Kuhn's method, 267

Meat-poisoning, 167
Meckel's diverticulum,
Mensuration, 220
Mercurial ulcerations,
Metabolism, 274
Metastases in intestina
Meteorism, 397; etiol
  matology a
  treatment o
Micrococci of intestine
Mineral springs, 316
Mixed intestinal juices
  tion of, 53
Mucin in feces, 242
Mucus in feces, 234, 2
Muscle-fibers in feces,
Myomata, 723

N.

Neoplasms: intestinal,
  (see Can
  benign, 7
  7
  7
  gastric s
  tinal, 260
Nervous diarrhea, 352
Neurasthenia intestinali

O.

Obstipation, 356; autoin
  consequenc
  sis of, 377;
  ology of, 3
  386; gymn
  dropathic p
  injections i
  in, 385; m
  mechanical
  methods, 39

P.

Packs, hot and cold, 344
Palpation: method of, 216; and percussion of abdomen, 214
Pancreas mistaken for tumor, 215
Pancreatic juice, 40; ferments of, 41; disturbances in secretion of, 128; results of absence of, 129; alkalinity of, 417; effects of suppression of, 417
  icterus, 130; calculi, 236, 246
Pancreatitis, hemorrhagic, 420
Papilloma, 716
Peptic duodenal ulcer, 594
Percussion, 219
Pericolitis, 504
Peristalsis of intestines, 84; effects of drugs and toxins on, 104; effects of intestinal gases on, 104; influence of nerves on, 89; influence of disturbance of circulation on, 94; influence of medicinal agents on, 102; mechanism of, 90; objects of, 85; pendulum movements of, 86; pathological, 95; rolling movement of, 80; visible through abdominal walls, 209
Peristaltic movement, proper, 85
Peyer's patches, 20
Pfeiffer reaction, 174
Phenol, 249
Phlegmonous enteritis, 477
Polycholia, 411
Proctitis, acute, 452
Proteids: assimilable and nonassimilable, 69; relative amounts absorbed in different forms, 69
Proteolysis, arrested, 114
Proteolytic ferments in colon and rectum, 308
Proteus vulgaris, 165; chemical products of, 166; pathogenic properties of, 166
Protozoa of intestines, 191
Psychic gastric juice, 44
Puncture, exploratory, 221; technic of, 222
Purgatives and laxatives, 320; modes of action, 320
Pus in feces, 236, 240, 656

R.

Rectal alimentation, 301; digestion, physiology of, 305; enemata, preparation of, 311; feeding, technic of, 312; feeding, indications for, 313
Rectum, cancer of, 692
Round duodenal ulcer, 594

S.

Salt, rôle of, 117
Sarcolactic acid formed in meats, 106
Sarcoma and lymphosarcoma, 701; age in, 707; diagnosis, 707; duration of, 707; etiology, 705; symptomatology, 706; tumor in, 707; treatment, 708
Scurvy, intestinal ulcers in, 651
Scybala, 231
Secretion, relation of gastric to duodenal, 52
Sedatives, 330
Sigmoid flexure, 31
Sigmoiditis, 453, 504; definition, 504; etiology, 505; diagnosis, 507; pathology, 507; treatment, 507
  chronic, 508; diagnosis, 510; treatment, 509
Skatol, 249
Small intestine, 17; blood supply of, 21; glands of, 20; lumen of, 18; length of, 17; lacteals and lymphatics of, 22; mucous membrane of, 18; nerves of, 21
Solitary glands, 20
Spices and relishes, importance in nutrition and digestion, 116
Spirillum: of Asiatic cholera, 183; morphology, 183; cultural characteristics, 184; pathogenesis, 185; accidental human infection by, 185
  of Finkler-Prior, 187; differential diagnosis, 187
Splenic flexure, 31
Starch in feces, 238
Steapsin, 42
Stenoses: duodenal, gastric chemistry in, 260; jejunal, gastric chemistry in, 262
Stercoral diarrhea, 351; ulcer, 628

Stool: in intestinal cancer, 688; in intestinal ulcer, 655; mucoid masses like sago in, 658
Stool sieve, Boas', 230
Streptococci in milk, 165
Streptococcus: pyogenes, 164; enteritis, due to, 165
Stricture in intestinal cancer, 687
Subcutaneous alimentation, 301, 315
Succorrhœa pancreatica, 414
Succus entericus: disturbances in secretion of, 124; results of decrease of secretion of, 124

T.

Teniæ coli, 27
Test-meals, duodenal, 414
Thrombotic ulcers, 620
Tonics, intestinal, 329
Toxic ulcerations, 652
Trypsin, 41
Tubercle bacilli in stool, 657
Tubercular ulcer, 631
Tuberculosis, intestinal, diet for, 300
Tumors: palpation of, 218; fecal, 218
Tympanites, 398
Typhoid bacillus, 148
Typhoid fever: macroscopic test for, 177; microscopic test for, 177
Typhotoxin, 181

U.

Ulceration: amyloid, 624; due to acute infectious diseases, 629; follicular, 627; toxic, 652
Ulcerative colitis, 479
Ulcer: intestinal: condition of stool in, 655; diarrhea in, 654; diet for, 663; prognosis, 662; pain in, 659; symptomatology and diagnosis, 654; treatment, 662; urine in, 660
Ulcer: peptic or round duodenal, 594; age, 598; complications of, 614; diagnosis of, 606; etiology, 594; frequency of, 603; latency of, 605; location of, 595; prognosis of, 613; progress of, 613; pathological anatomy, 598; sex, 595; symptomatology, 600; symptoms of perforation in, 612; treatment, 615
embolic and thrombotic, 620; occurring in neuritis, 623
catarrhal intestinal, 626; stercoral or decubital, 628
tubercular, 631; etiology and pathogenesis of, 638; histology of, 643
syphilitic, 647; due to scurvy, 651; due to gout, 651; due to, leukemia, 652; mercurial, 653; free HCl in, 259
Urine, 250; amount of, 251; in intestinal ulcer, 660

V.

Valvulæ conniventes, 18
Vegetable diet, indigestible matter in, 133
Vermiform appendix, 29
Villi, 19

W.

Widal reaction, 174, 177; culture used for, 180; technic, 180

X.

X-rays in diagnosis of intestinal disease, 213

Y.

Yeasts, 162

L860 Hemmeter, John C
H489 Diseases of the intestines 4643
1901-2
v.1

NAME	DATE DUE
Boardman -	ch. March 9 1915
W. T. Dawson .	AUG [illegible] 1923 SEP 8 '24